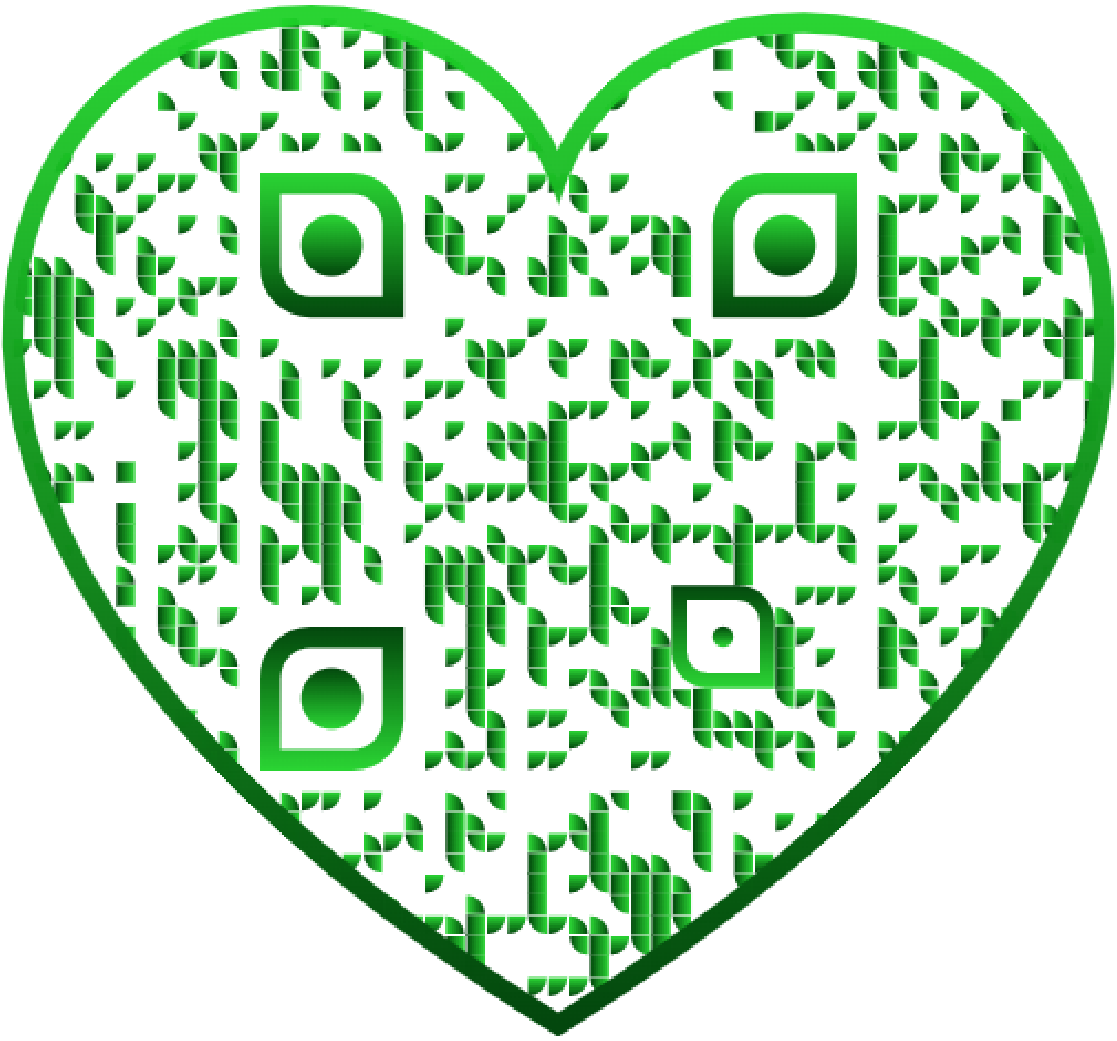
بسم الله الرحمن الرحيم



Introduction

Dr Abdelwassa El Mekhlafi; MD

What is dermatology

❈ Dermatology is the study of both normal and abnormal skin and associated structures such as hair, nails, and oral and genital mucous membranes.

Why is dermatology important?

❈Skin diseases are very common, affecting up to a third of the population at any one time.
❈ Skin diseases have serious impacts on life. They can cause physical damage, embarrassment, and social and occupational restrictions.
❈ Chronic skin diseases may cause financial constraints with repeated sick leave.
❈ Some skin conditions can be life-threatening

dermatological history

Presenting complaint: Nature , site and duration of problem

History of presenting complaint :

- Initial appearance and evolution of lesion
- Symptoms: (particularly itch and pain) Aggravating and relieving factors
- Previous and current treatments (effective or not)
- Recent contact, stressful events, illness and travel
- History of sunburn and Skin type

Past medical history: -History of atopy i.e. asthma, allergic rhinitis, eczema
-History of skin cancer and suspicious skin lesions

Family history :Family history of skin disease.

Social history :Occupation (including skin contacts at work) Improvement of lesions when away from work

Medication and allergies: Regular, recent and over-the-counter medications

Impact on quality of life :Impact of skin condition and concerns

Examining the skin

There are four important principles in performing a good examination of the skin:

INSPECT, DESCRIBE, PALPATE and SYSTEMATIC CHECK

-INSPECT *in general :* General observation, Site and number of lesion(s)

If multiple, pattern of distribution and configuration

DESCRIBE: *the individual lesion* SCAM

-Size (the widest diameter), Shape

-Colour

-Associated secondary change

-Morphology, Margin (border)

**If the lesion is pigmented, remember* ABCD

(the presence of any of these features increase the likelihood of melanoma):

-Asymmetry (lack of mirror image in any of the four quadrants)

-Irregular Border -Two or more Colours within the lesion -Diameter

PALPATE *the individual lesion* Surface

- Consistency - Mobility - Tenderness - Temperature

SYSTEMATIC CHECK Examine the nails, scalp, hair & mucous membranes, General examination of all systems

-Pruritus : (Itching) an unpleasant cutaneous sensation which provokes the desire to scratch or rub the skin.

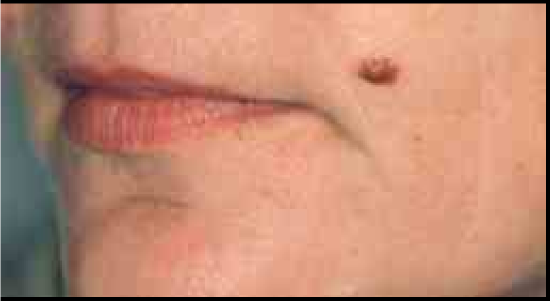
-Rash : An eruption: the term rash is used to describe the presence of many lesions.

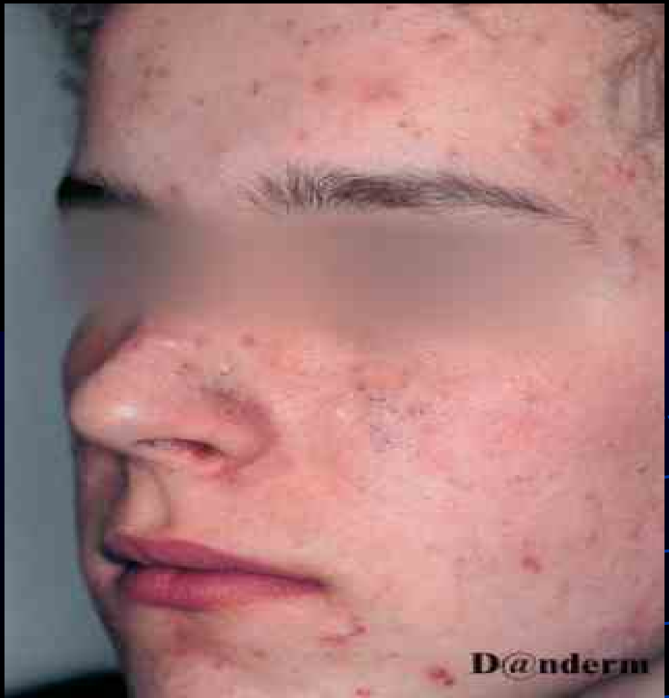
-Lesion: An area of altered skin

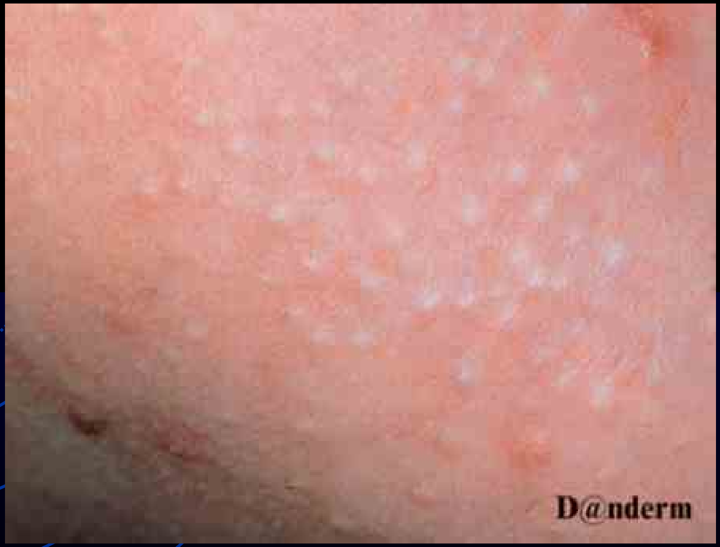
-**Pain:** may be deep and burning

-Naevus : A localised malformation of tissue structures

-Comedone : A plug in a sebaceous follicle containing altered sebum, bacteria and cellular debris; can present as either open (blackheads) or closed (whiteheads)

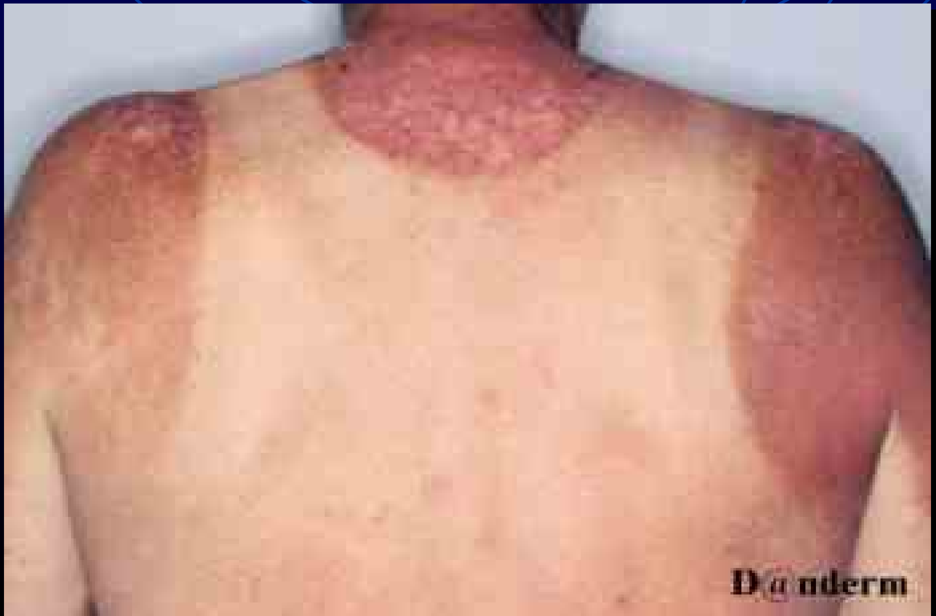


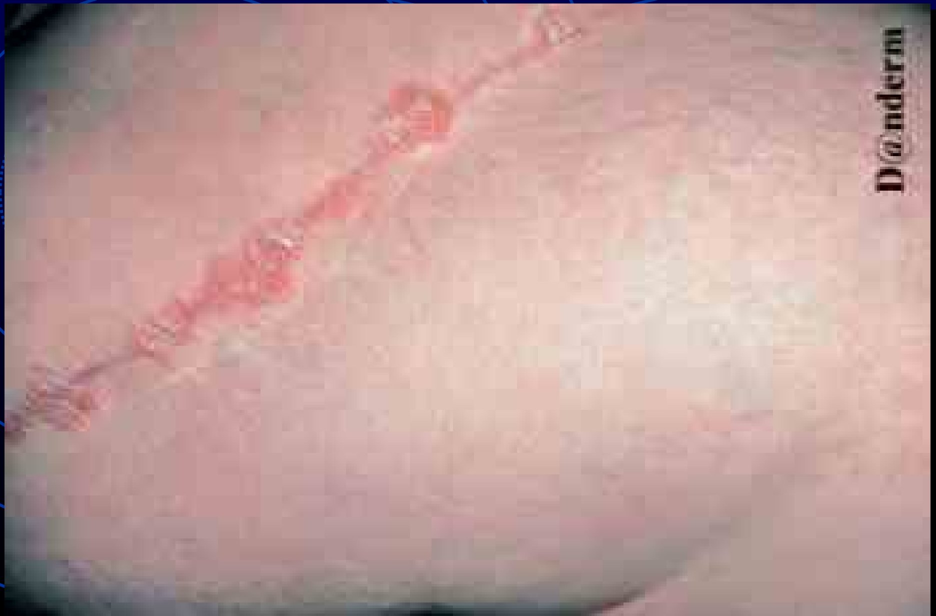




Distribution (the pattern of spread of lesions)

-Generalised All over the body
-Widespread Extensive
-Localised Restricted to one area of skin only
-Flexural: Body folds i.e. groin, neck, behind ears, popliteal and antecubital fossa
-Extensor Knees, elbows, shins
-Pressure areas Sacrum, buttocks, ankles, heels
-Dermatome An area of skin supplied by a single spinal nerve
-Photosensitive Affects sun-exposed areas such as face, neck and back of hands
-Kebner phenomenon A linear eruption arising at site of trauma it is seen in (ps; lichen P; vitiligo; plane wart, molluscum contagiosum,…..).



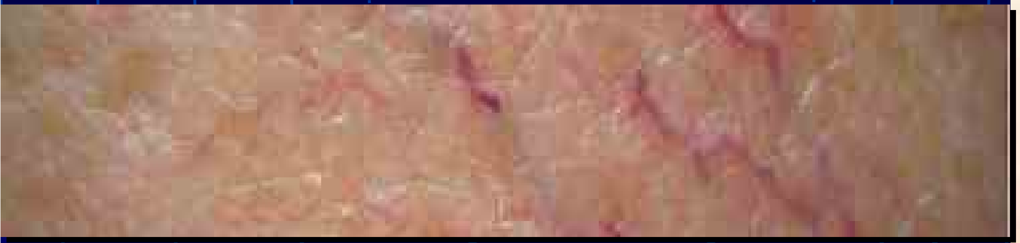


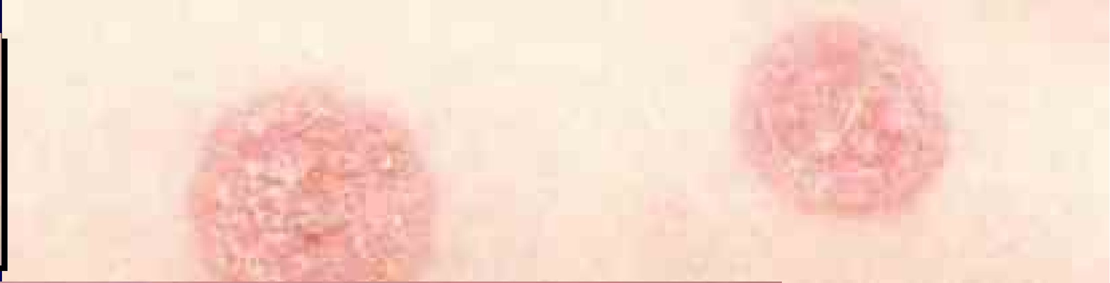
Configuration (the pattern or shape of grouped lesions)

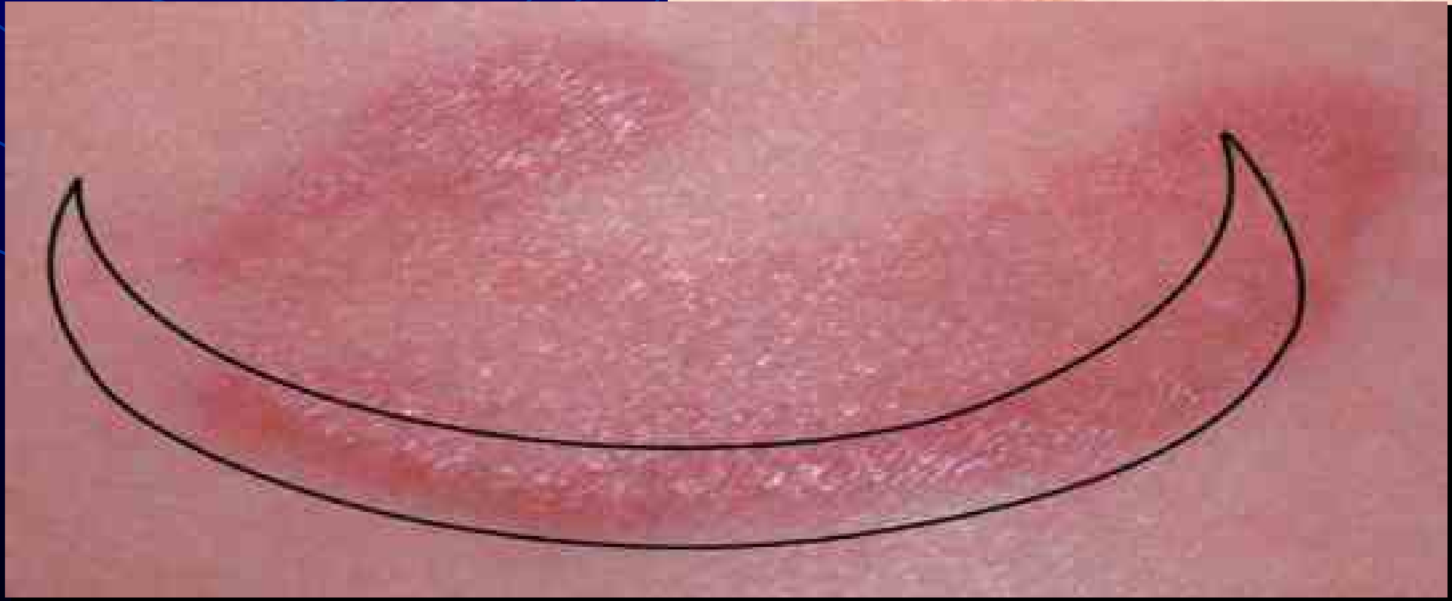
- Discrete: Individual lesions separated from each other
- Confluent: Lesions merging together
- Linear: In a line
- Target: Concentric rings (like a dartboard)
- Annular :Like a circle or ring
- Discoid /Nummular :A coin-shaped/round lesion

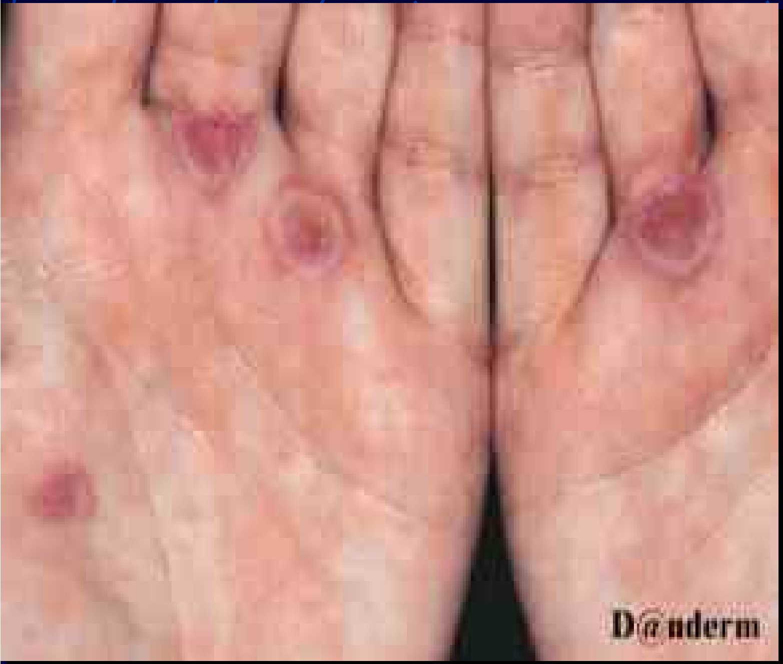
Arrangement of Lesions

arcuate	curved (just kidding)
Annular	ring-shaped
Serpiginous	wandering or twisting, like a snake
Herpetiform	tight clustering of lesions heaped on top of on another.
Dermatomal	lesions in a dermatome: shingles!

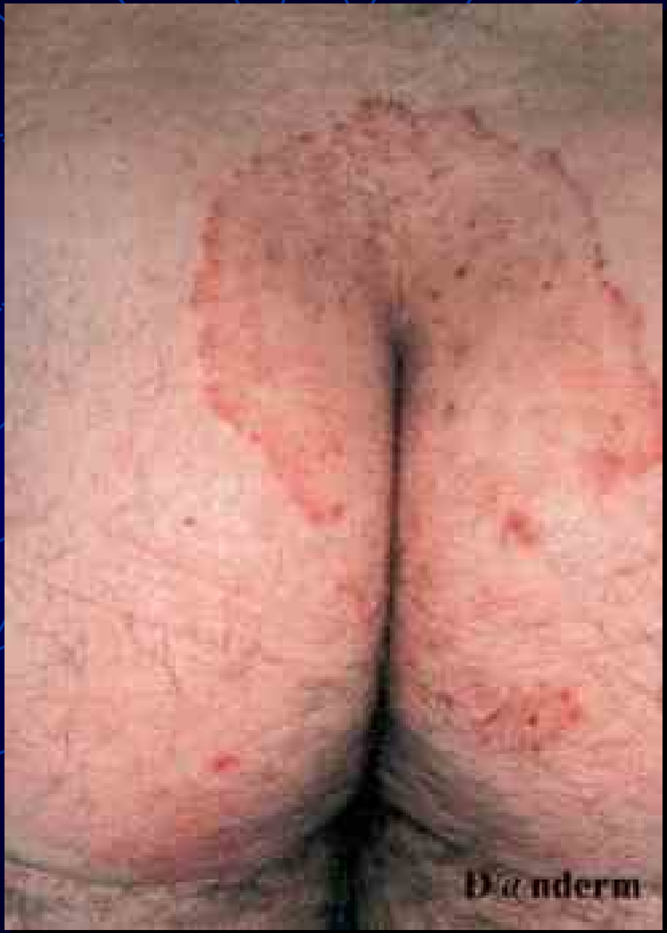




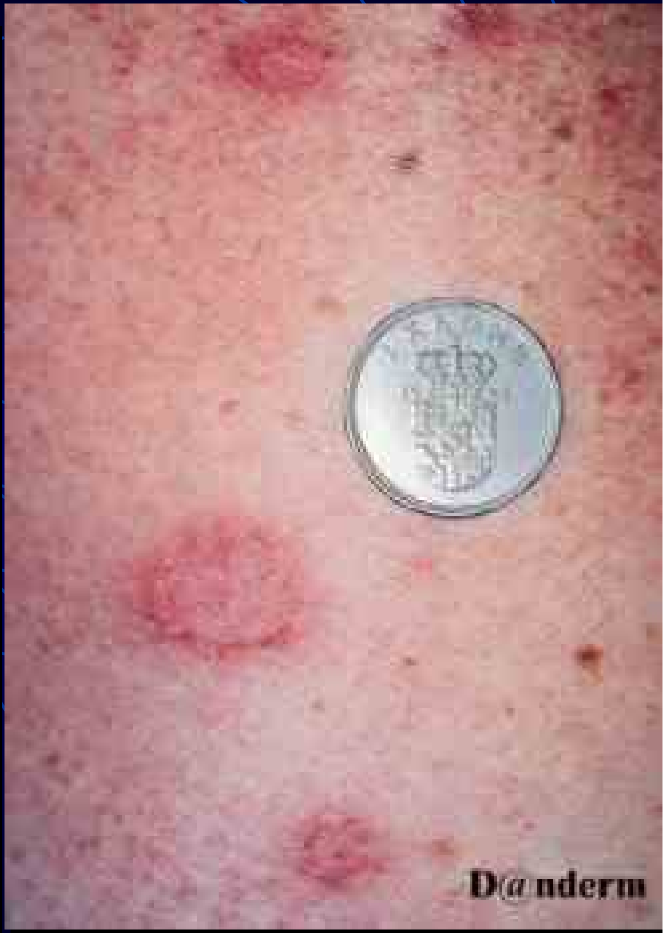




D@nderm



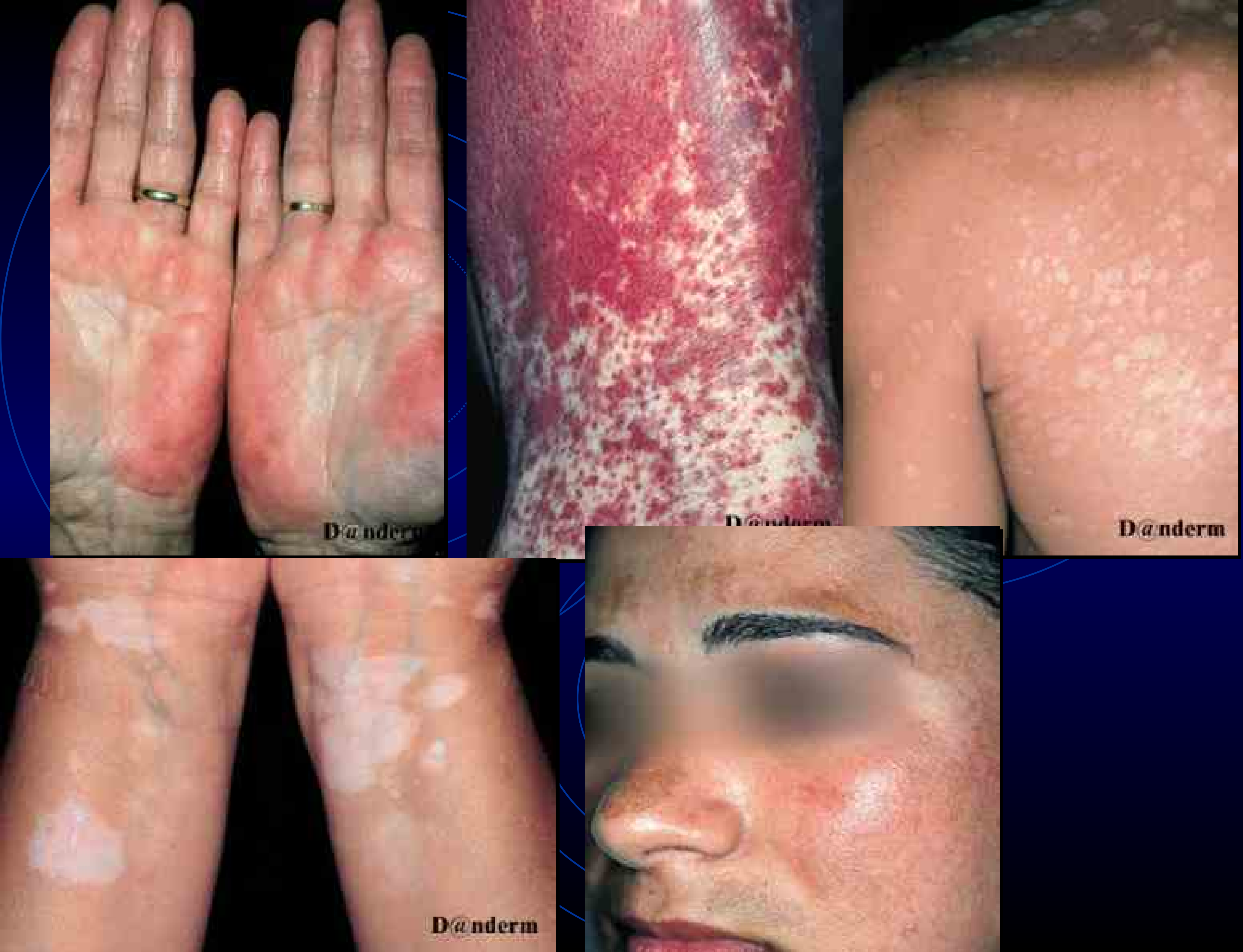
D@nderm



D@nderm

Colour

- Erythema: Redness (due to inflammation and vasodilatation) which blanches on pressure
- Purpura: Red or purple colour (due to bleeding into the skin or mucous membrane) which does not blanch on pressure – **petechiae** (small pinpoint macules) and **ecchymoses** (larger bruise-like patches)
- Hypo- pigmentation: Area(s) of paler skin
- De- pigmentation: White skin due to absence of melanin
- Hyper-pigmentation: Darker skin which may be due to various causes (e.g. post-inflammatory)
- Telengiectasia: Delated dermal vessels


D@nderm
D@nderm
D@nderm
D@nderm

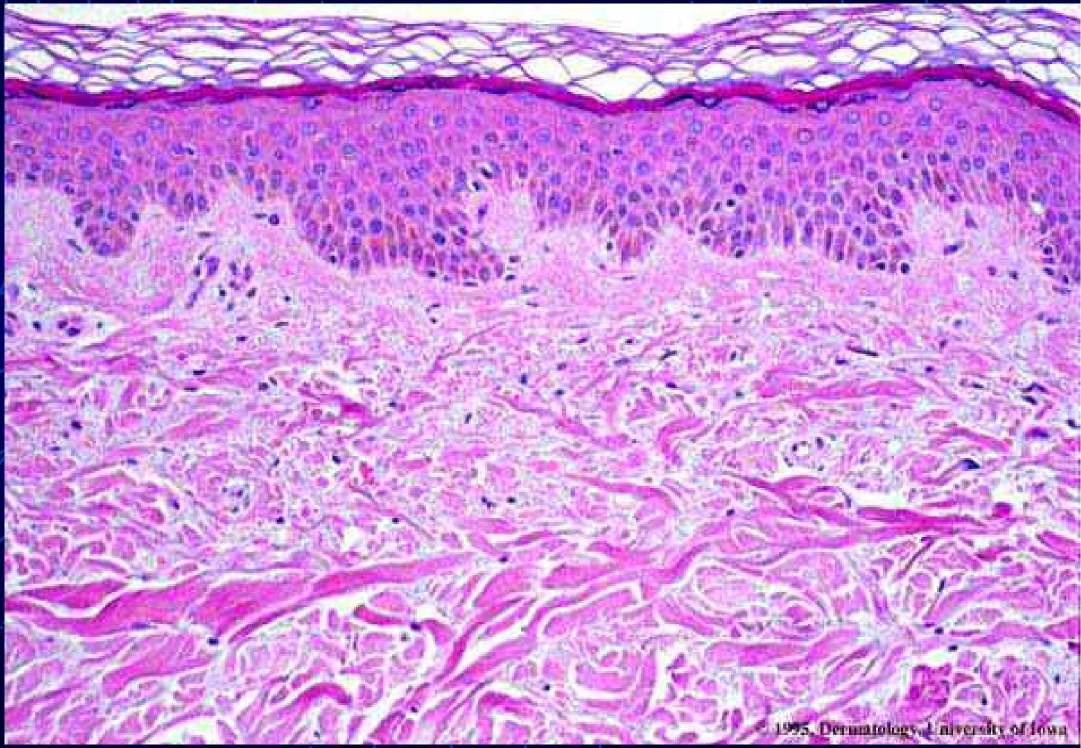
investigation

- Skin biopsy
- Immunofluoresence (direct/indirect)
- Wood,s light examination
- Mycology examination (KOH, fungal culture)
- Bacteriology examination (gram stain, bact. viral Culture,)
- Mite exam. To identify the burrow
- Blood and urine tests
- Dark field
- Cytology exam. Tzanck smear

Structure of the skin

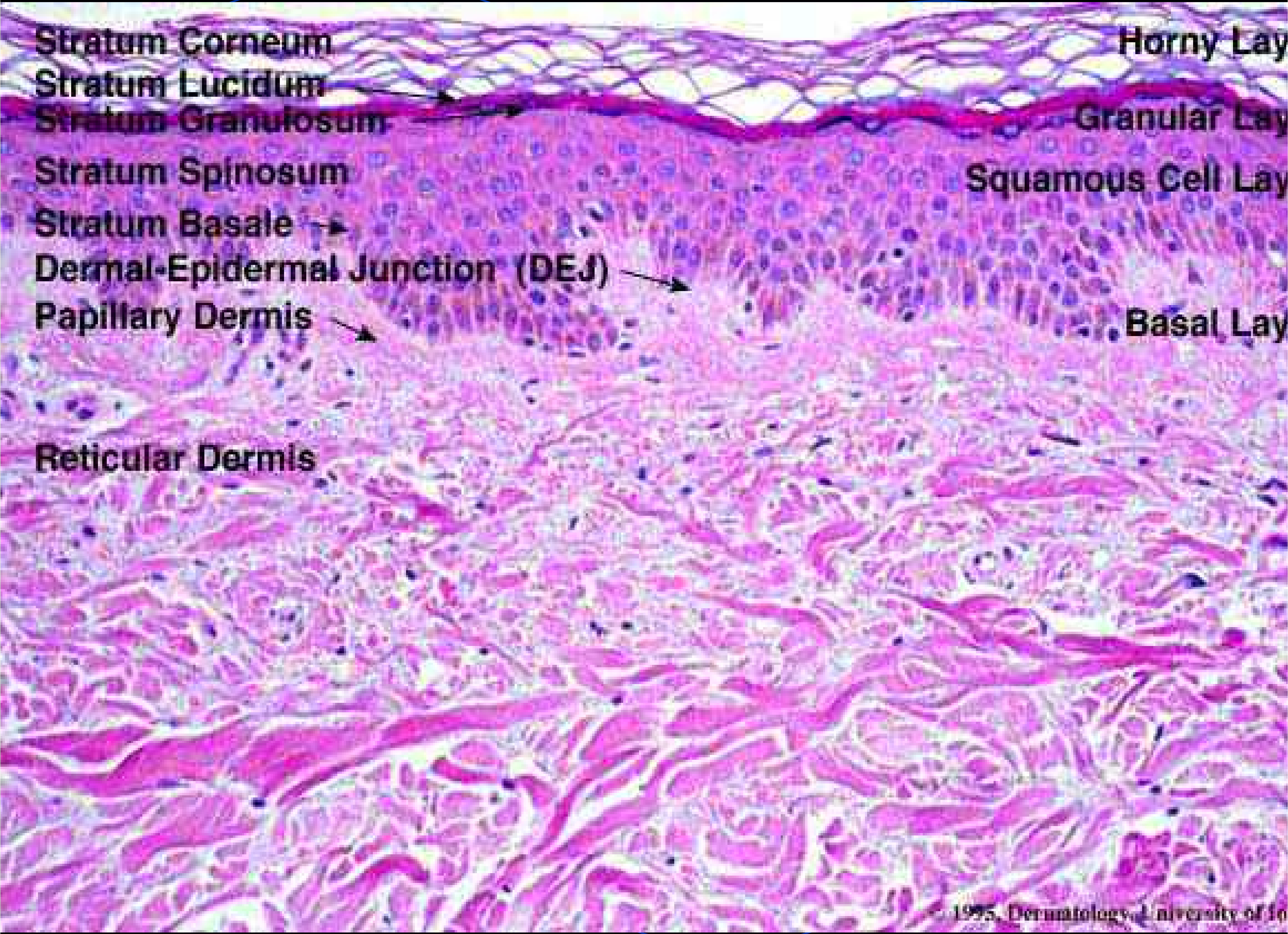
The skin is composed of 3 layers: epidermis, dermis and subcutaneous tissue.

- *Epidermis*
- The adult epidermis is composed of 3 basic cell types:
- keratinocytes, melanocytes & Langerhans cells.
- The ***keratinocyte***, or ***squamous cell***, is the principal cell of the epidermis.

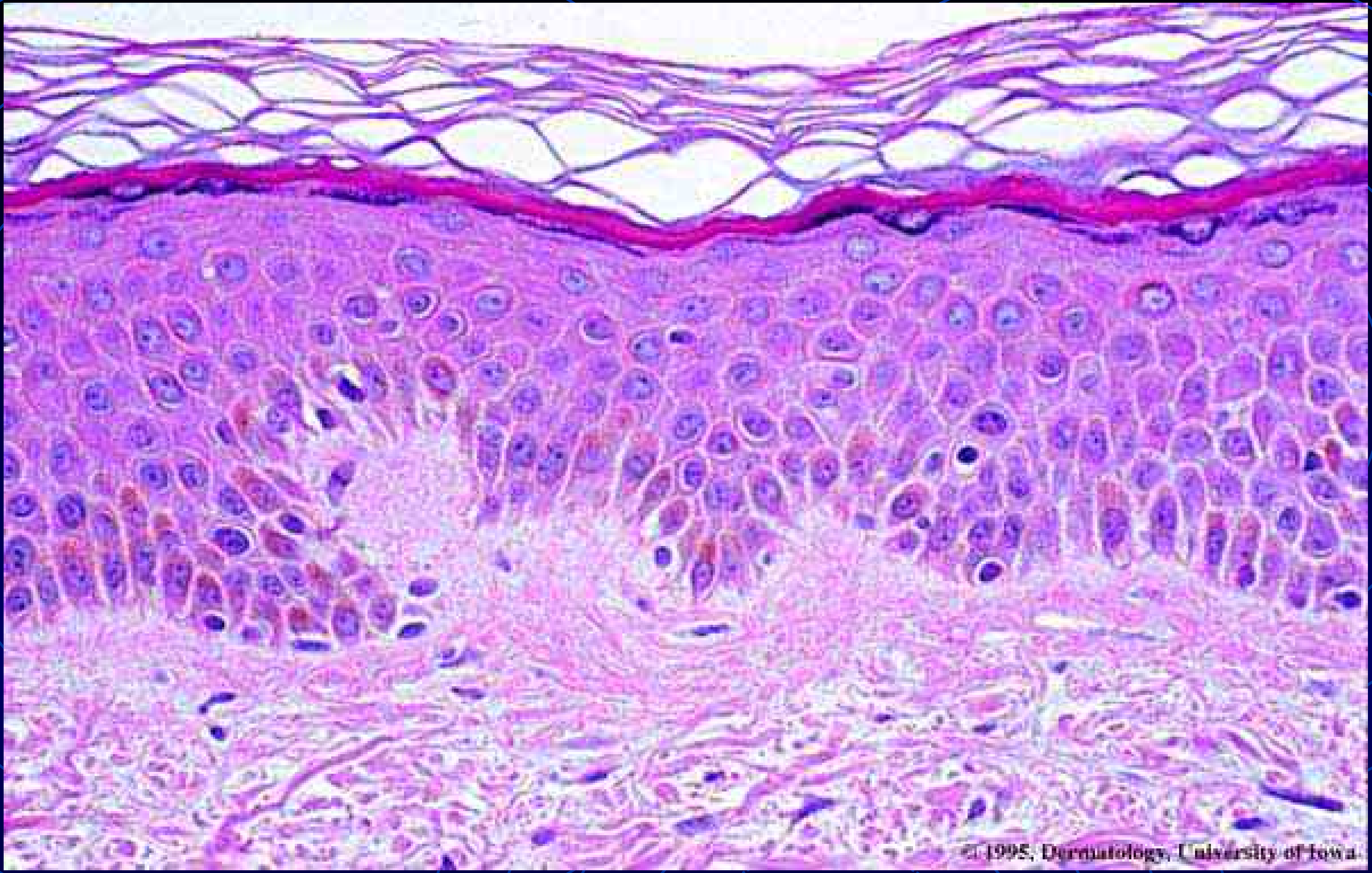

© 1995, Dermatology, University of Iowa

Epidermis

- The epidermis is divided into 4 main layers beginning with the innermost layer
- 1. Basal layer.
- 2. Spinous layer.
- 3. Granular layer.
- 4. Horny layer.

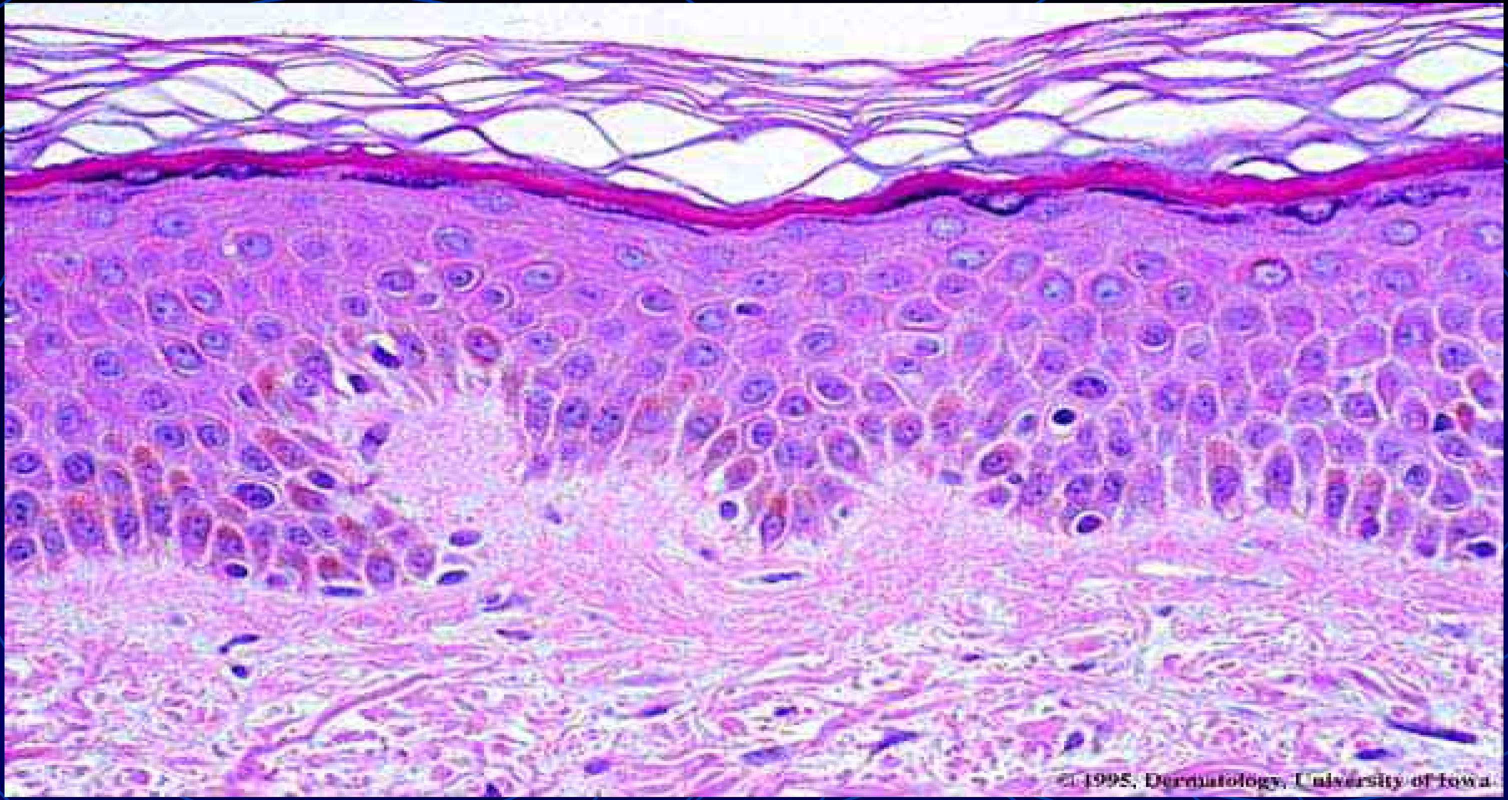

Stratum Corneum
Stratum Lucidum
Stratum Granulosum
Stratum Spinosum
Stratum Basale
Dermal-Epidermal Junction (DEJ)
Papillary Dermis
Reticular Dermis
Horny Lay
Granular Lay
Squamous Cell Lay
Basal Lay

1995, Dermatology, University of Io



© 1995, Dermatology, University of Iowa

- The ***melanocyte*** is the dendritic pigment-producing cell of the epidermis and found in the basal layer.



© 1995, Dermatology, University of Iowa

- The *Langerhans cell* is a dendritic cell present in the malpighian layer. Functionally, it is of the monocyte-macrophage lineage and originates in bone marrow. Langerhans cell plays a role in some immunological reactions

Dermis

- The dermis, which is bounded externally by its junction with the epidermis and internally by subcutaneous fat, contributes to 15-20% of the total weight of the human body. It largely consists of a supporting matrix or ground substance and connective tissue formed mainly of collagen and elastin. all of which will decrease with age.
- The predominant cellular elements in the dermis are *fibroblasts*. The dermis also contains nerve endings, sweat glands, sebaceous glands, hair follicles , blood vessels and lymphatics. The **papillary dermis** contains smaller blood vessels which supply oxygen, and nutrients to the lower epidermis.
- The thicker **reticular dermis** contains dense connective tissue, larger blood vessels, elastic fibres and bundles of collagen arranged in layers.
- Also within the reticular layer are the following key cell types:
- **- fibroblasts – a key cell involved in repairing tissue damage**
- **- mast cells – which are involved in fighting infection**
- **- lymphatic vessels – the lymphatic system is a key part of the body's defence against infection**
- **- epidermal appendages**
- **- ground substance – a gel-like substance that helps to support the cells within the dermis and provides structure to the area.**

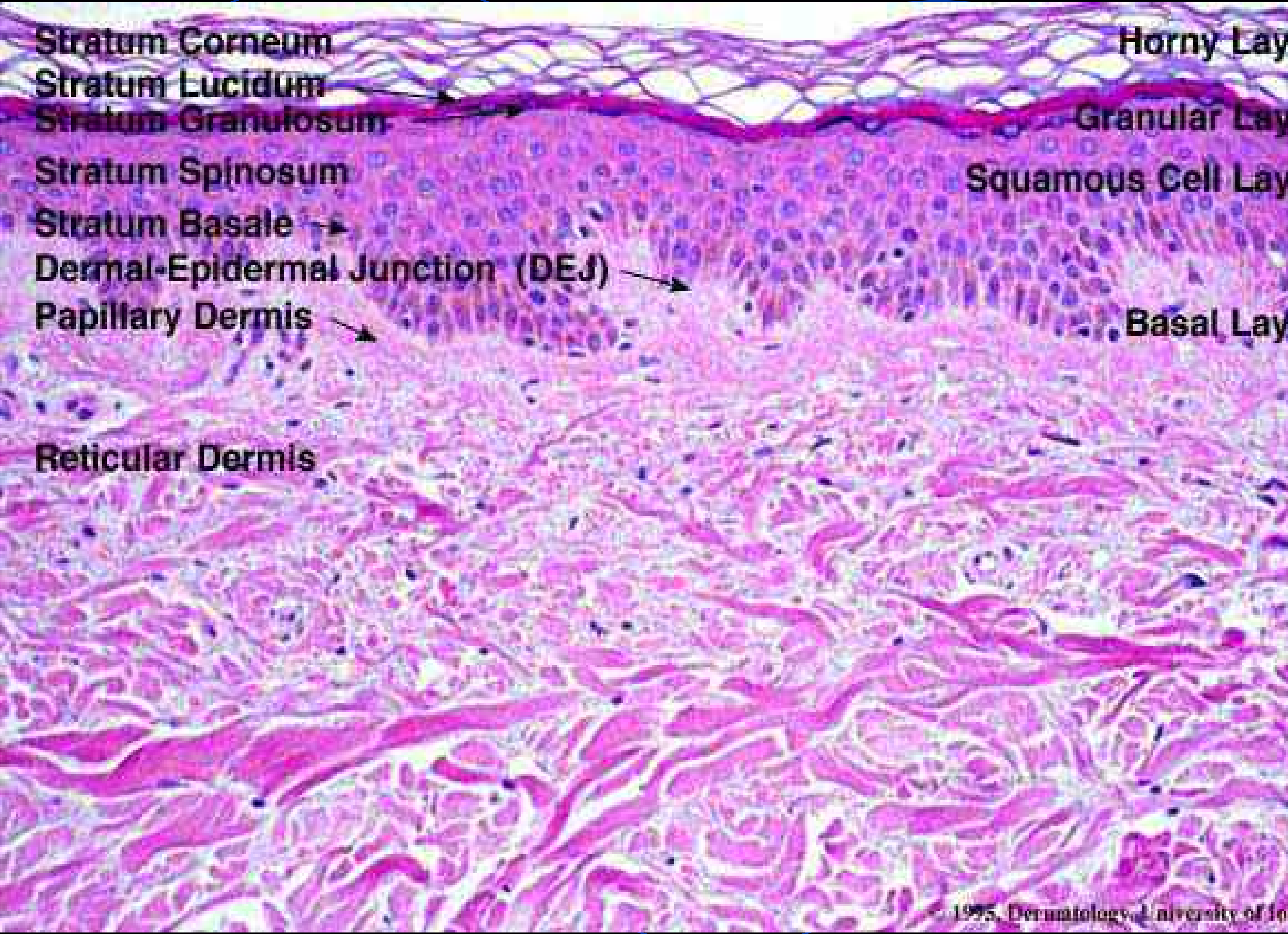
The dermo-epidermal junction

The junction of epidermis and dermis is formed by the basement membrane zone which is considered to be a semipermeable filter that permits exchange of cells and fluid between the epidermis and dermis. It further serves as a structural support for the epidermis and holds the epidermis and dermis together.

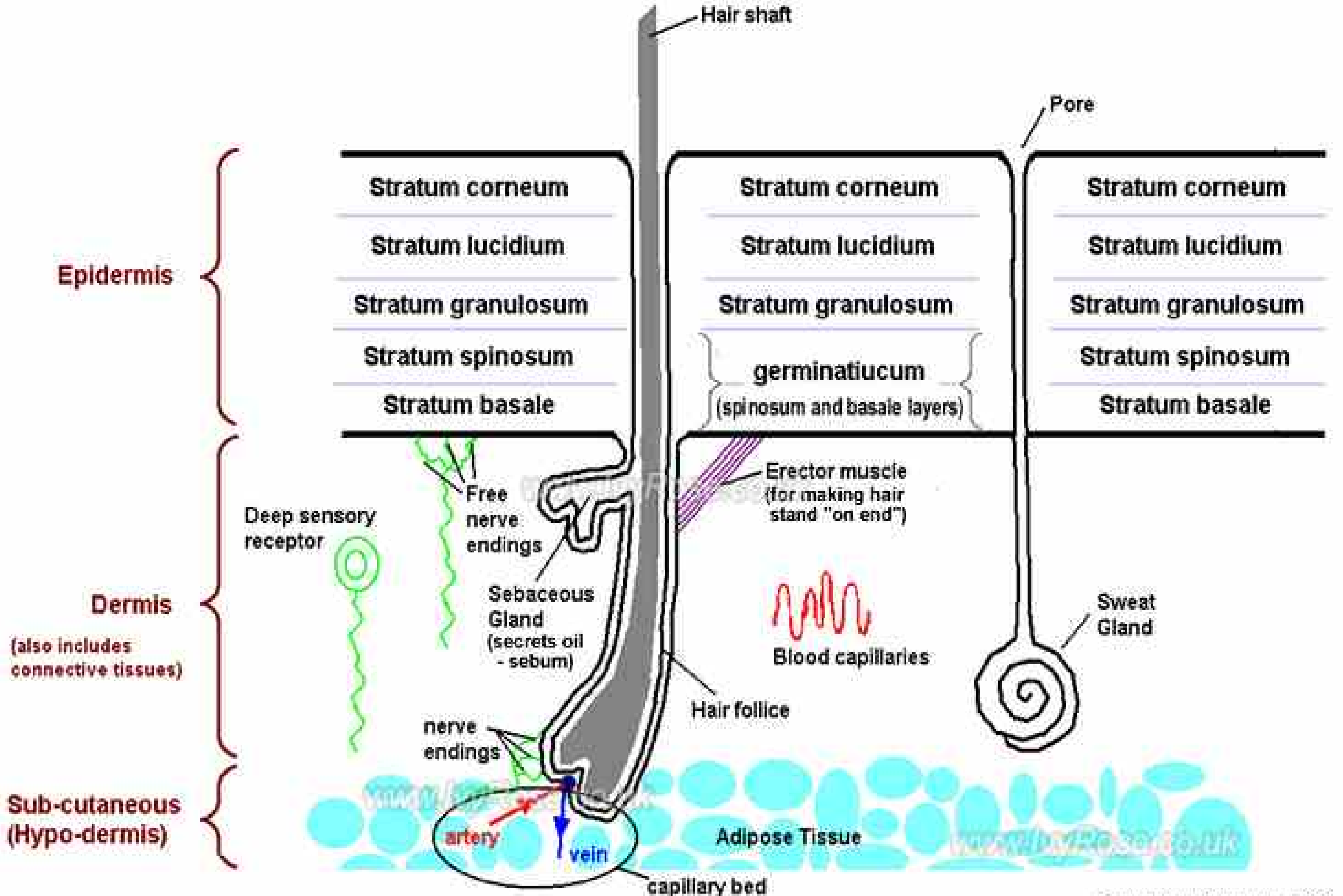
The epidermis and dermis interdigitate through downwards projections of the epidermis (**rete pegs**) and upwards projections of the dermis (**papillae**).

Subcutaneous tissue

Beneath the dermis lies the subcutaneous tissue which is composed of lobules of fat cells separated by fibrous septa and large blood vessels.


Stratum Corneum
Stratum Lucidum
Stratum Granulosum
Stratum Spinosum
Stratum Basale
Dermal-Epidermal Junction (DEJ)
Papillary Dermis
Reticular Dermis
Horny Lay
Granular Lay
Squamous Cell Lay
Basal Lay

1995, Dermatology, University of Io


Hair shaft
Pore
Epidermis
Stratum corneum
Stratum lucidium
Stratum granulosum
Stratum spinosum
Stratum basale
Stratum corneum
Stratum lucidium
Stratum granulosum
germinatiucum
(spinosum and basale layers)
Stratum corneum
Stratum lucidium
Stratum granulosum
Stratum spinosum
Stratum basale
Free nerve endings
Deep sensory receptor
Erector muscle (for making hair stand "on end")
Dermis
(also includes connective tissues)
Sebaceous Gland (secrets oil - sebum)
Blood capillaries
Sweat Gland
Hair follice
nerve endings
Sub-cutaneous (Hypo-dermis)
artery
vein
Adipose Tissue
capillary bed

Copyright IvyRose Ltd., 2003

Basic skin lesions

A- Primary lesions

These are the original lesions of the skin diseases.

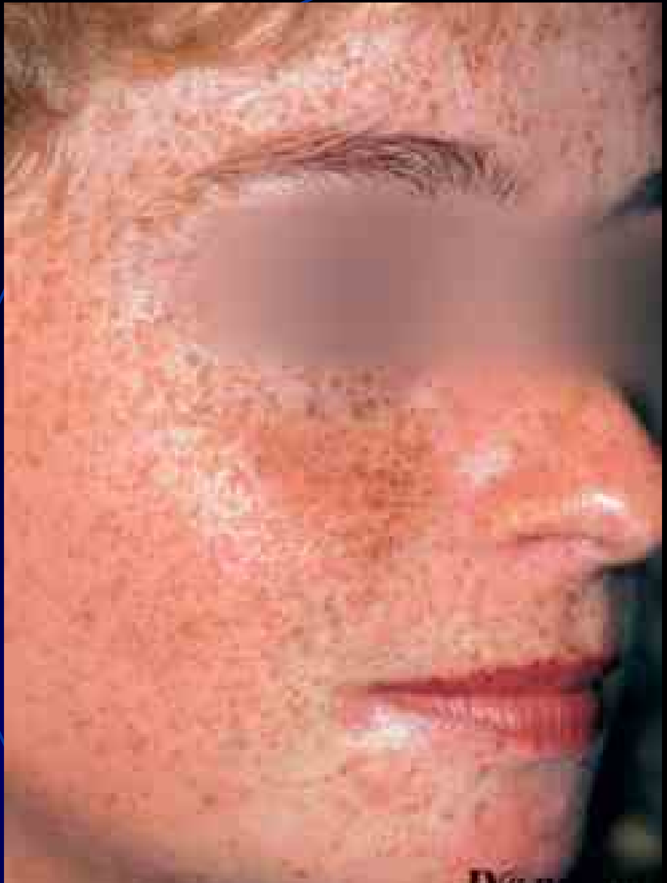
Macule

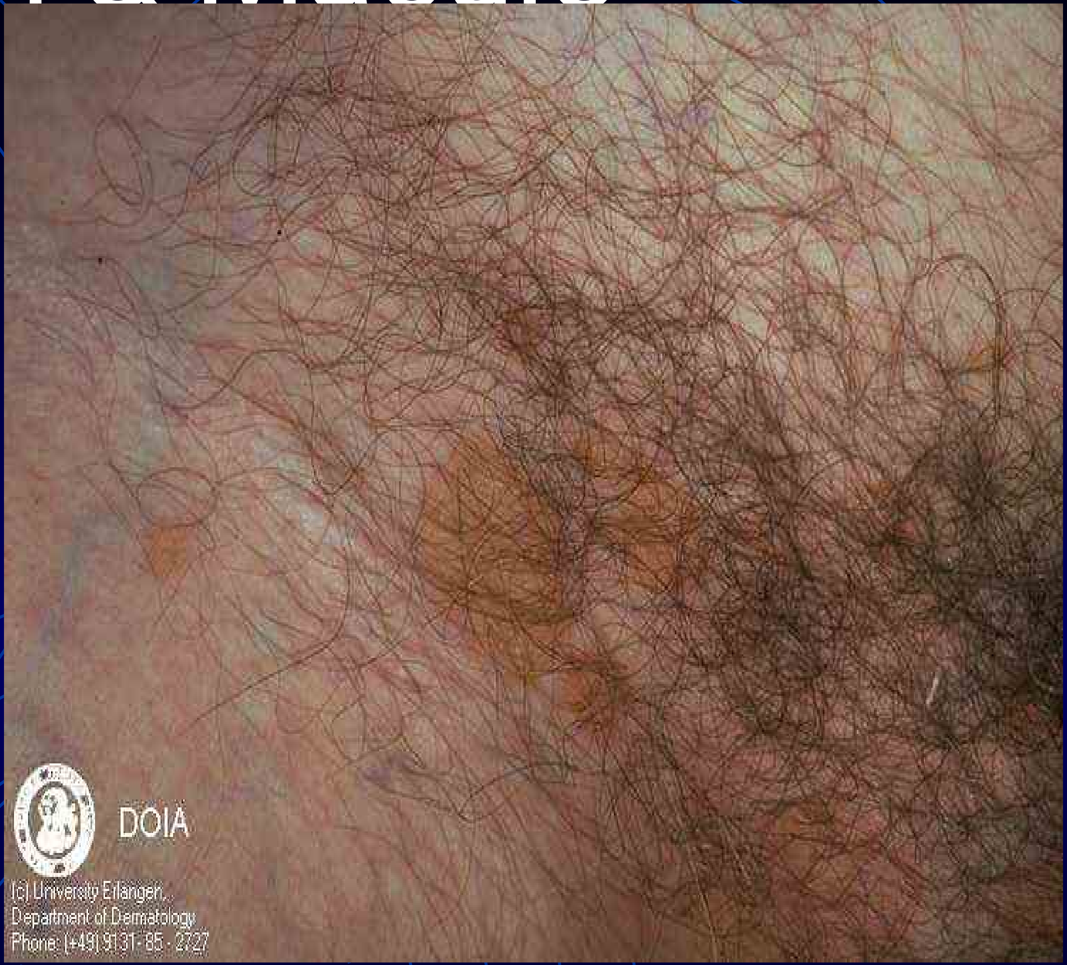
A circumscribed alteration in the color or texture of the skin less than 1 cm in diameter.

Patch

A macule larger than 1 cm in diameter.

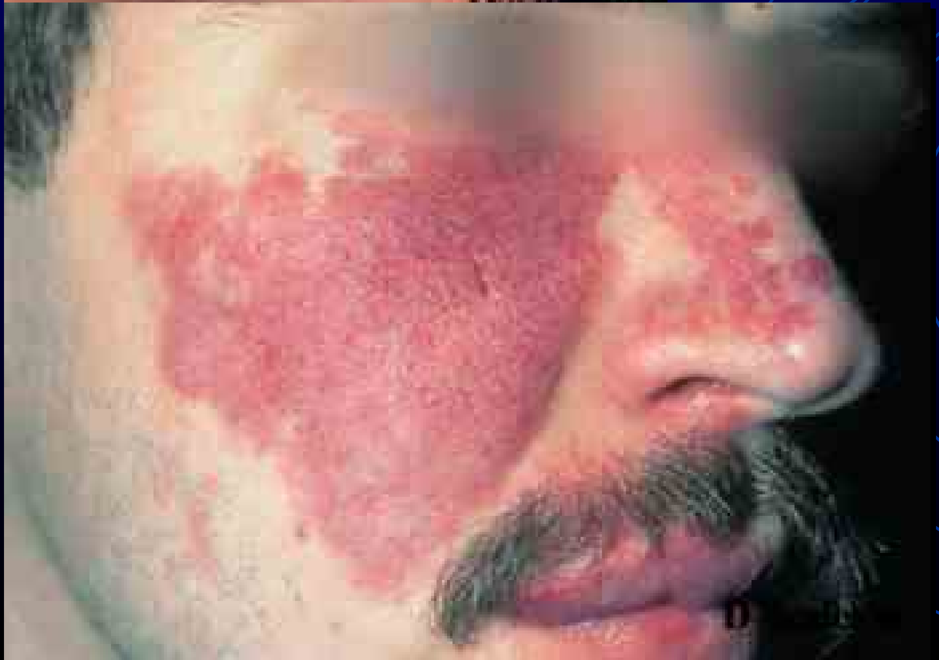
Patch & Macule





DOIA

(c) University Erlangen,
Department of Dermatology
Phone: (+49) 9131-85-2727



Basic lesions
A- Primary lesions

Papule

A circumscribed palpable elevation less than 0.5 cm in diameter.

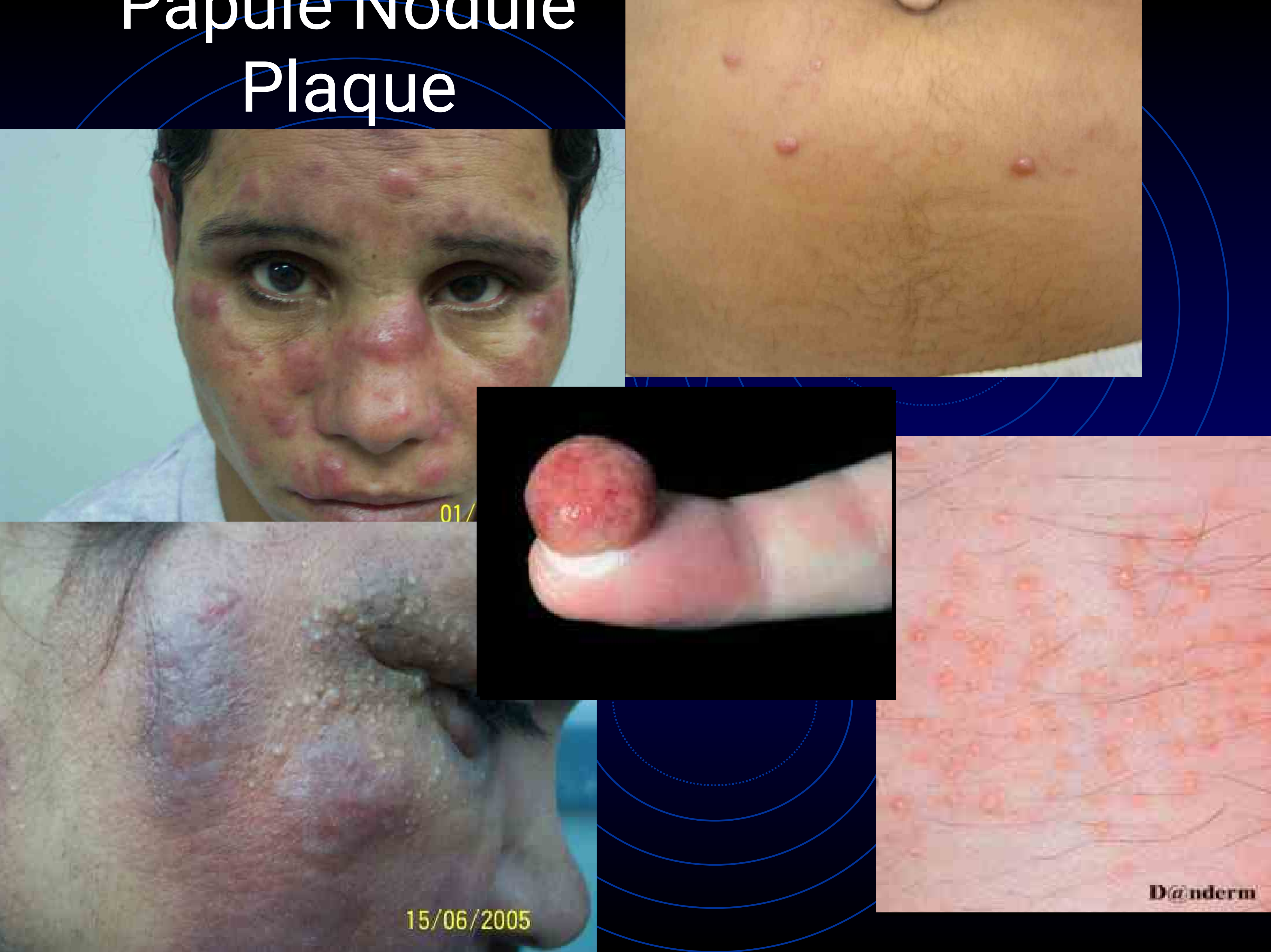
Nodule

A solid mass in the skin similar to the papule but more than 0.5 cm in diameter.

Plaque

An elevated area of skin 2 cm or more in diameter.

Papule Nodule Plaque


01/
15/06/2005
D@nderm

Basic lesions
A- Primary lesions

Vesicle (blister)

A fluid-filled lesion less than 0.5 cm in diameter.

Bulla (large blister)

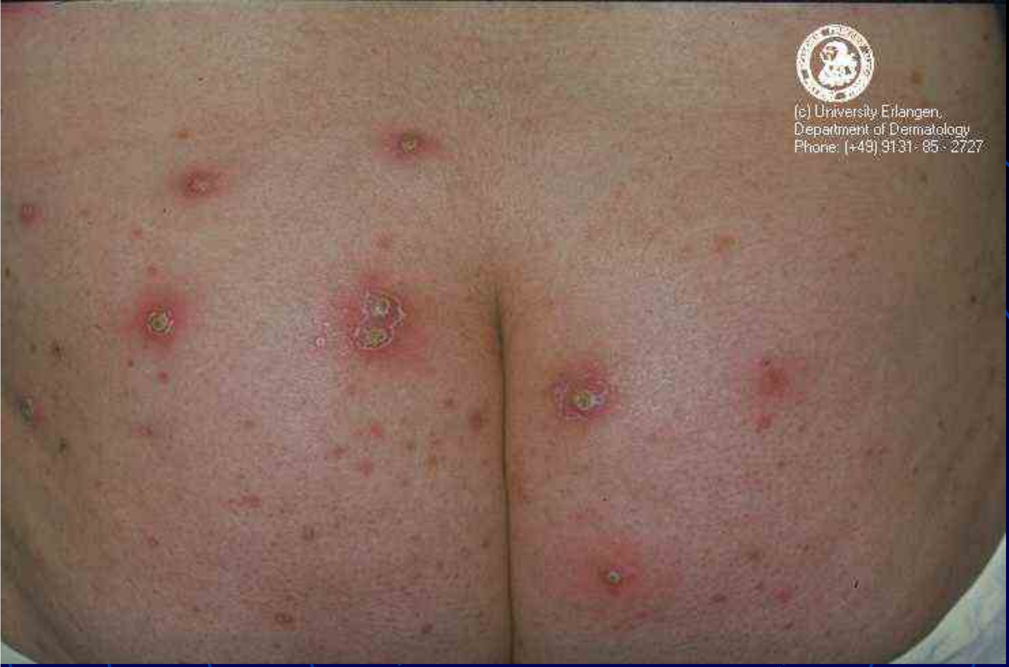
A fluid-filled lesion larger than 0.5 cm in diameter.

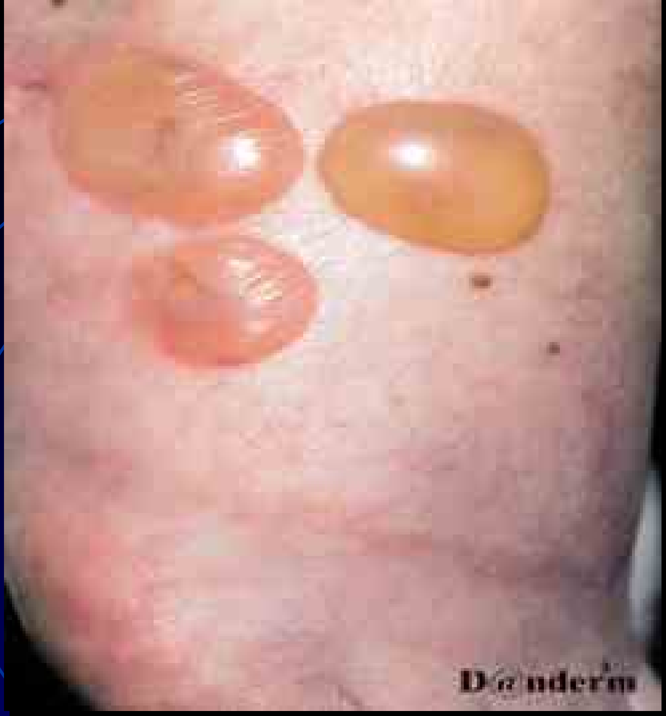
Pustule

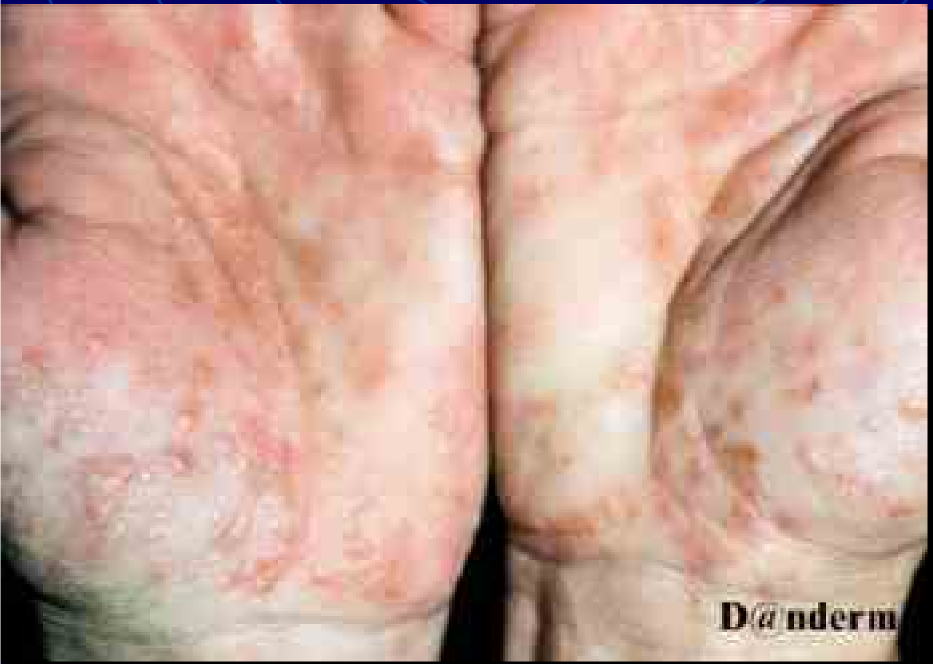
Small elevation of the skin similar to the vesicle but containing purulent material.

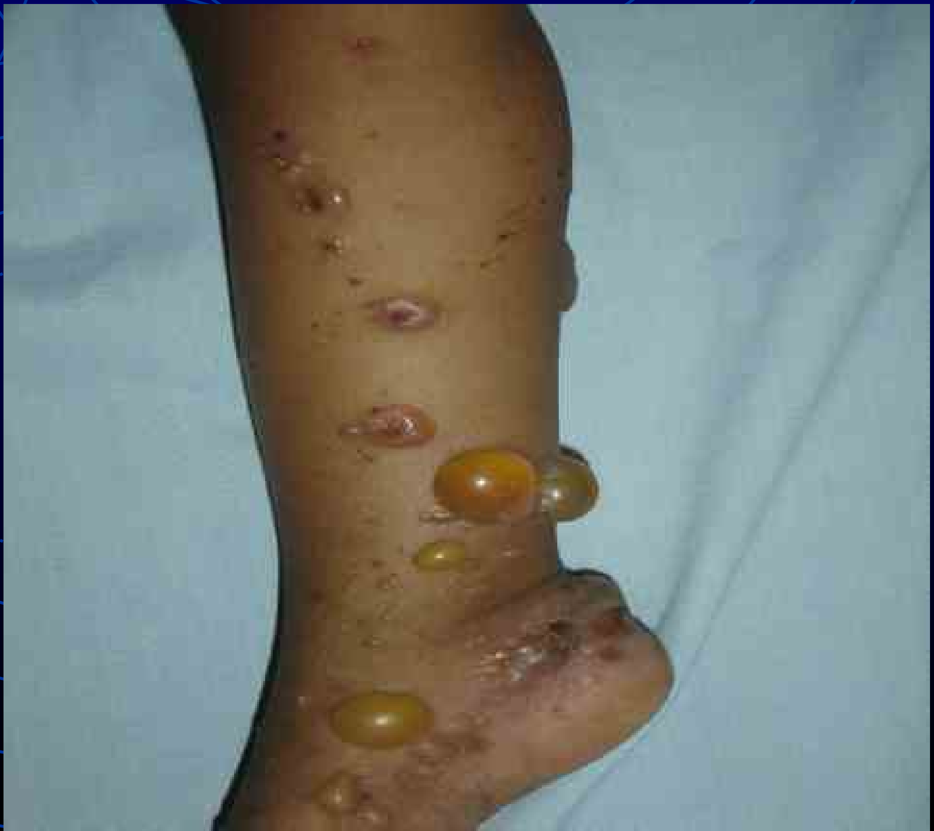
Abscess

Localised accumulation of pus in the dermis or subcutaneous tissues


(c) University Erlangen,
Department of Dermatology
Phone: (+49) 9131 - 85 - 2727


D@nderm


D@nderm



Basic lesions
A- Primary lesions

Wheal (weal).

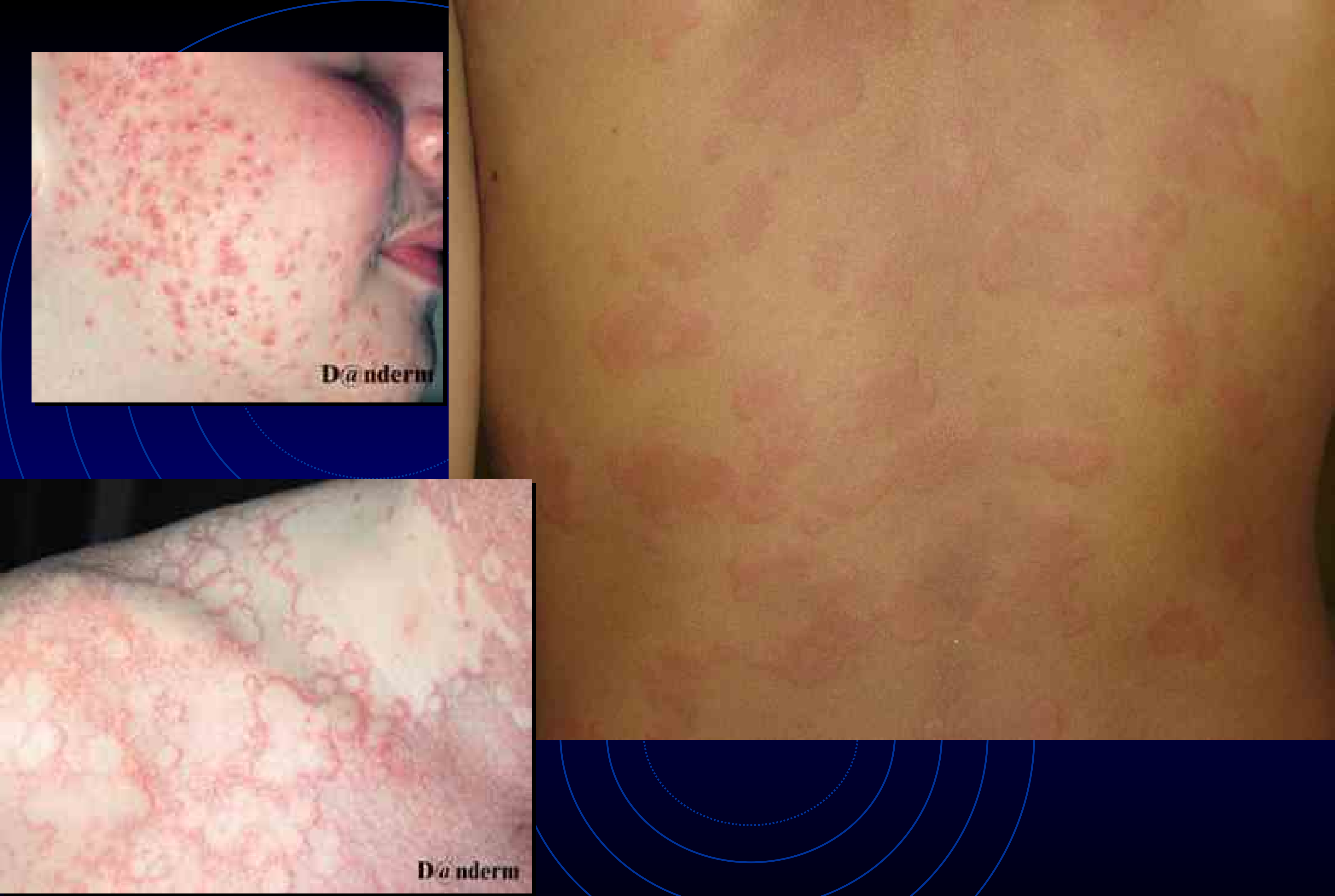
Evanescent, edematous, elevations of the skin of various sizes. It is the characteristic lesion of urticaria.

Comedone :

A plug in a sebaceous follicle containing altered sebum, bacteria and cellular debris; can present as either open (blackheads) or closed (whiteheads)

Burrow

A small tunnel in the skin, linear, ‘S’ shaped, 3-5 mm in length and found in patients with scabies


D@nderm
D@nderm

Basic lesions

- Secondary lesions

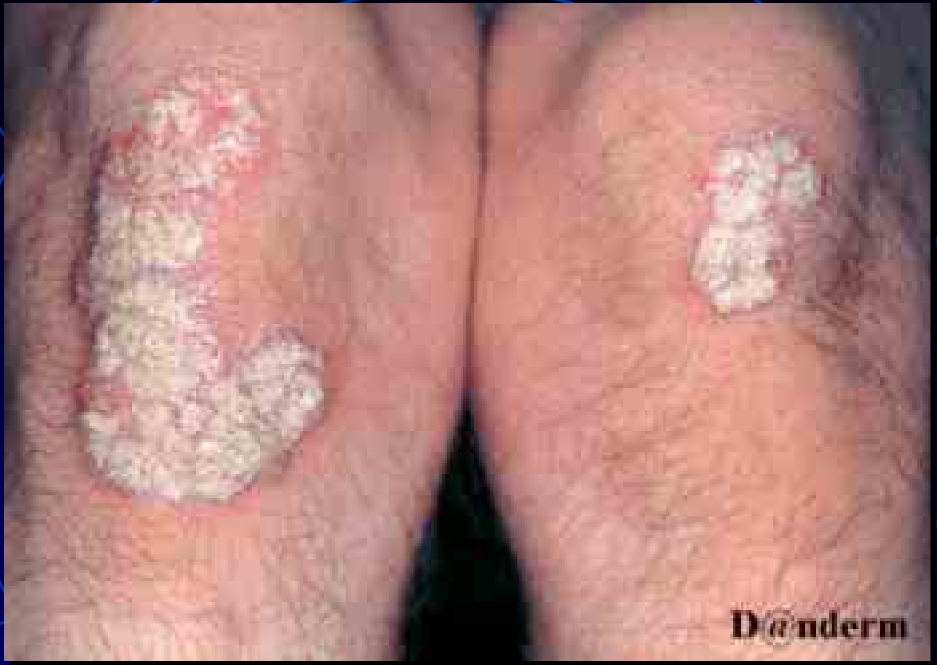
These have developed from modification of primary lesions by regression, or may be caused by external force such as scratching, infection or trauma.

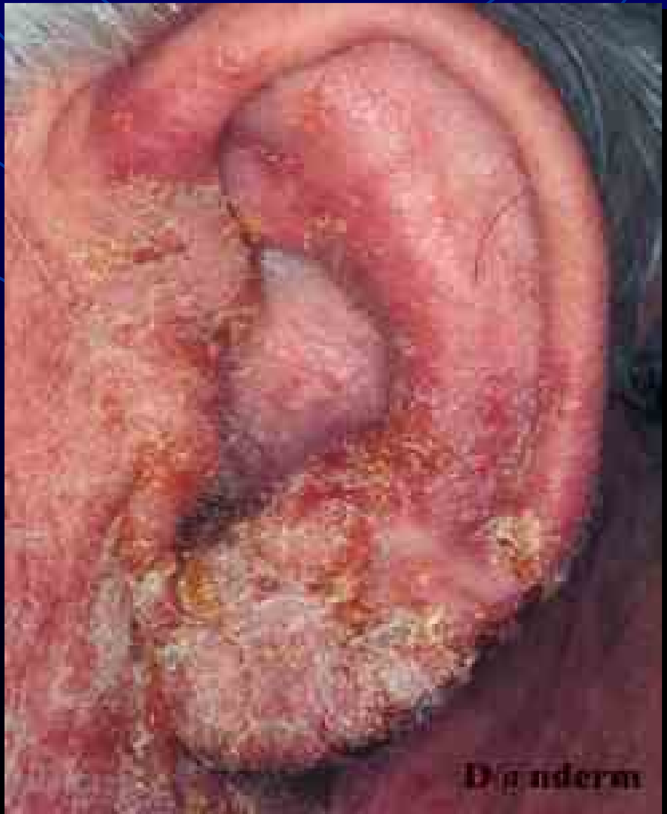
Scales

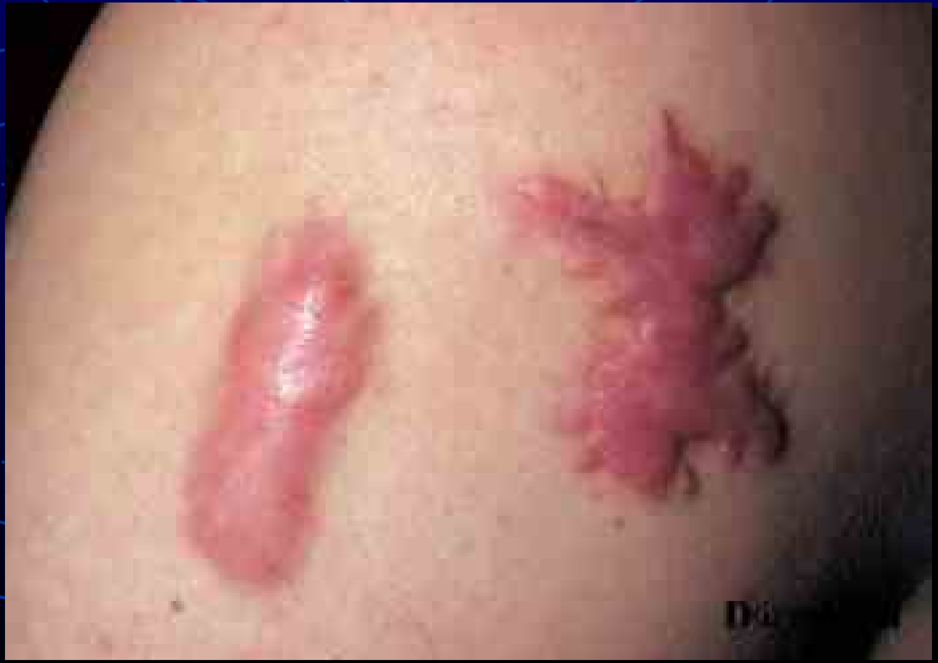
Dry or greasy laminated masses of keratin, may be primary in some diseases (e.g. psoriasis) or secondary (e.g. desquamation in scarlet fever).

Crusts

Consist of dried serum and other exudates.


D@nderm


D@nderm



Basic lesions

Secondary lesions

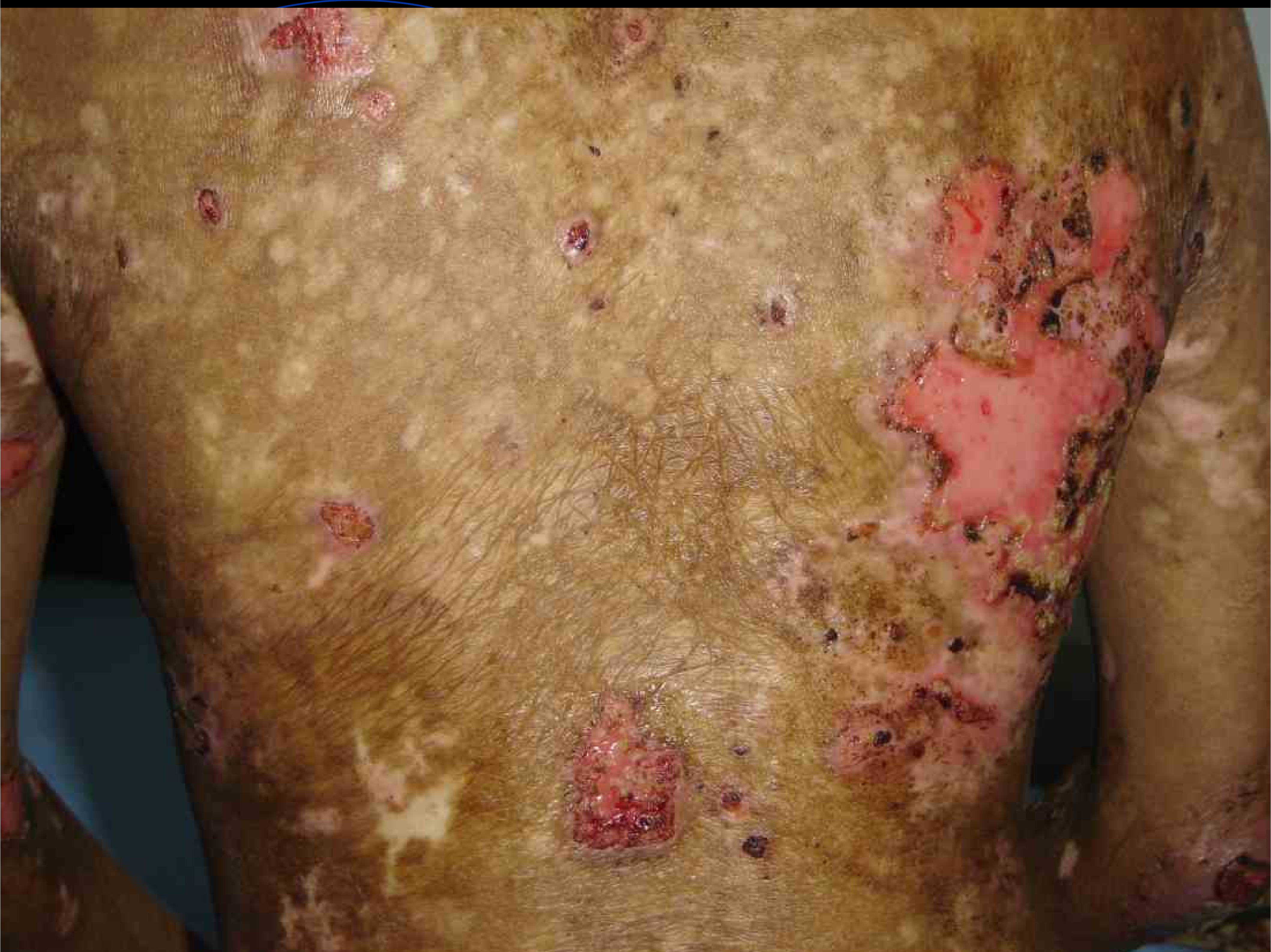
Erosion: A loss of epidermis which heals without scarring.

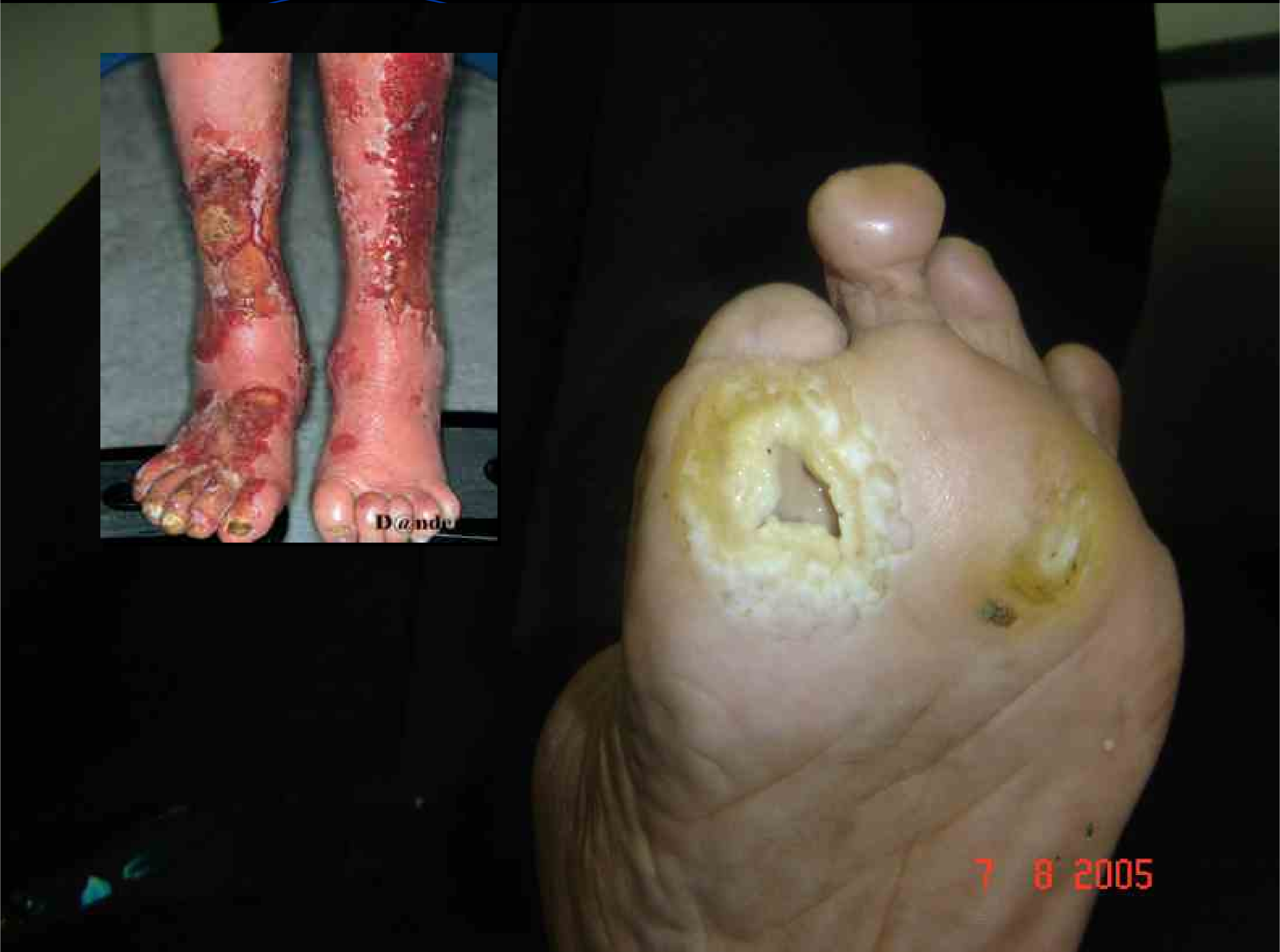
Ulcer: Complete loss of epidermis and dermis which heals with scarring.

Fissure: A linear gap or slit in the skin surface.

Scar: New fibrous tissue which occurs post-wound healing. It may be atrophic (thinning), or hypertrophic (hyperproliferation within wound boundary).

Keloid: hyperproliferation beyond wound boundary.




D@ndu
7 8 2005

Basic lesions

Secondary lesions

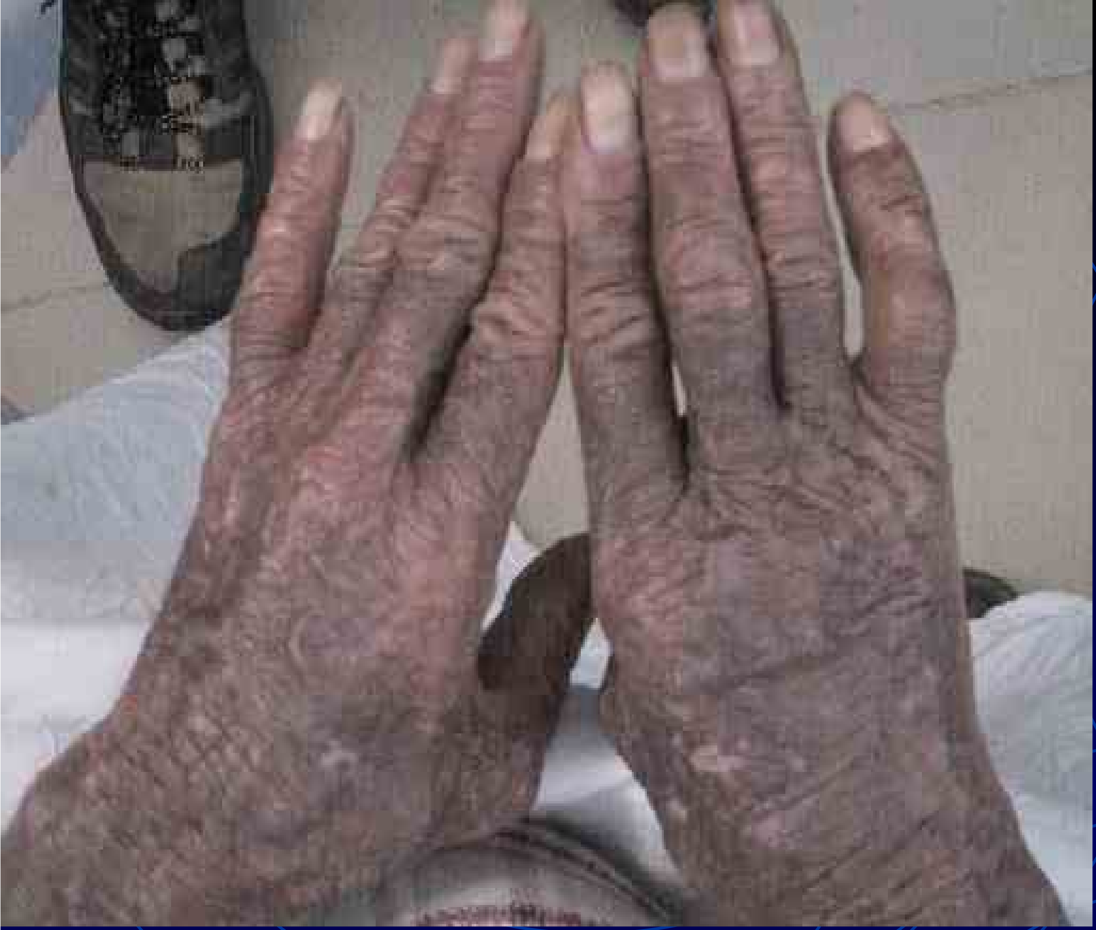
Excoriations (scratch marks)

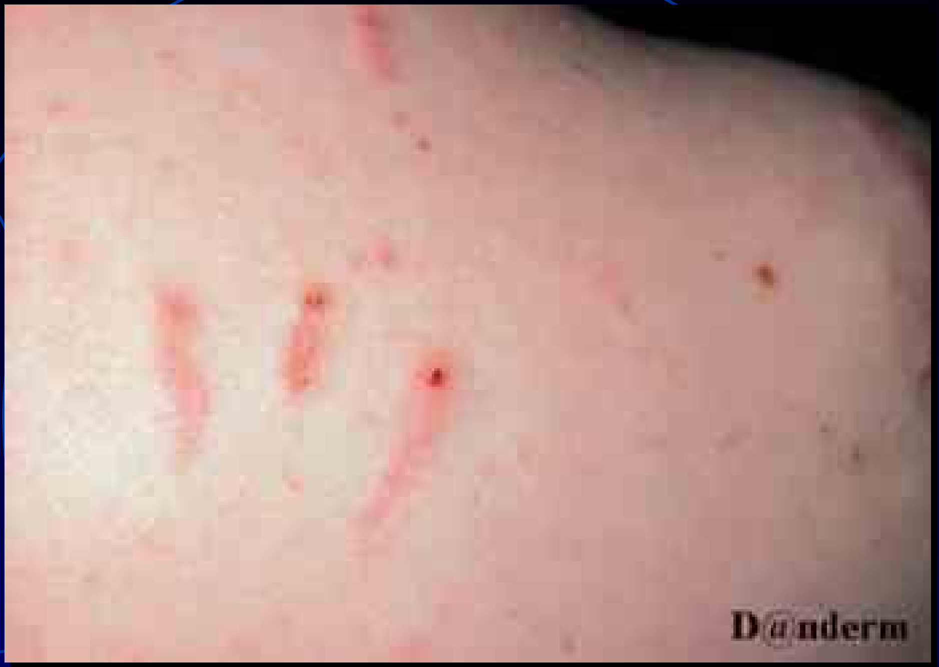
Loss of skin substance caused by scratching with the fingernails to relieve itching.

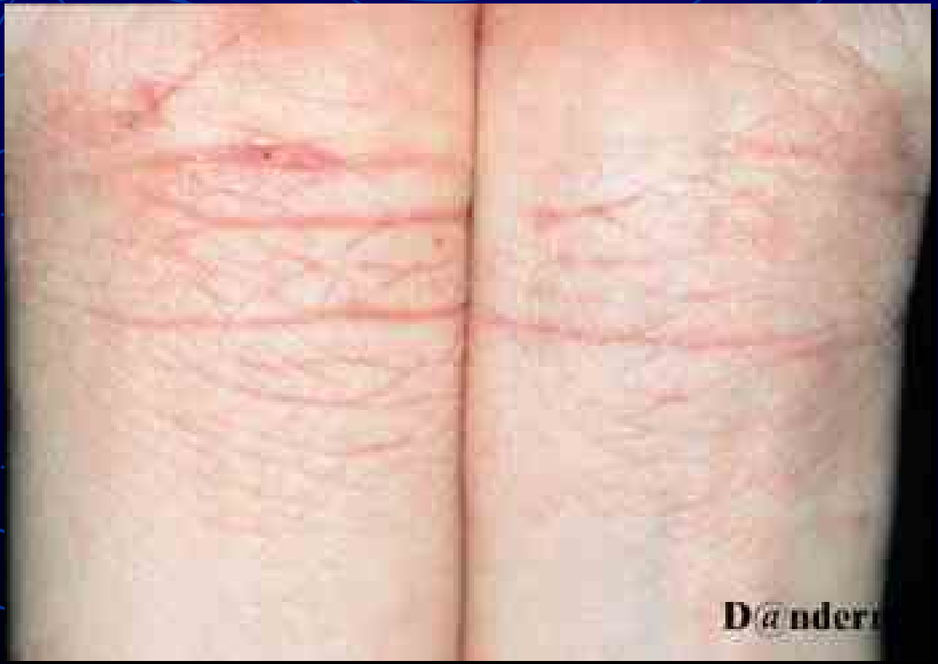
Lichenification

A pattern of cutaneous response to repeated rubbing or scratching and characterized clinically by thickened skin, with accentuation of the surface markings.

Atrophy: thinning of the epidermis, dermis or both




D@nderm


D@nder

Pathological terms

-**Hyperplasia:** Increase in the number of cells

-**Hyperkeratosis:** Thickening of the stratum corneum

- **Orthokeratosis :** Hyperkeratosis without parakeratosis

-**Parakeratosis:** retention Flattened keratinocyte nuclei within the stratum corneum

- **Acanthosis:** Thickened epidermis (increase in keratinocyte population of spinous layer)

-**Spongiosis:** Intercellular oedema between keratinocytes)

-**Acantholysis:** the loss of cohesion between epidermal keratinocytes

-**Dyskeratosis:** Faulty and premature keratinization of individual keratinocytes.

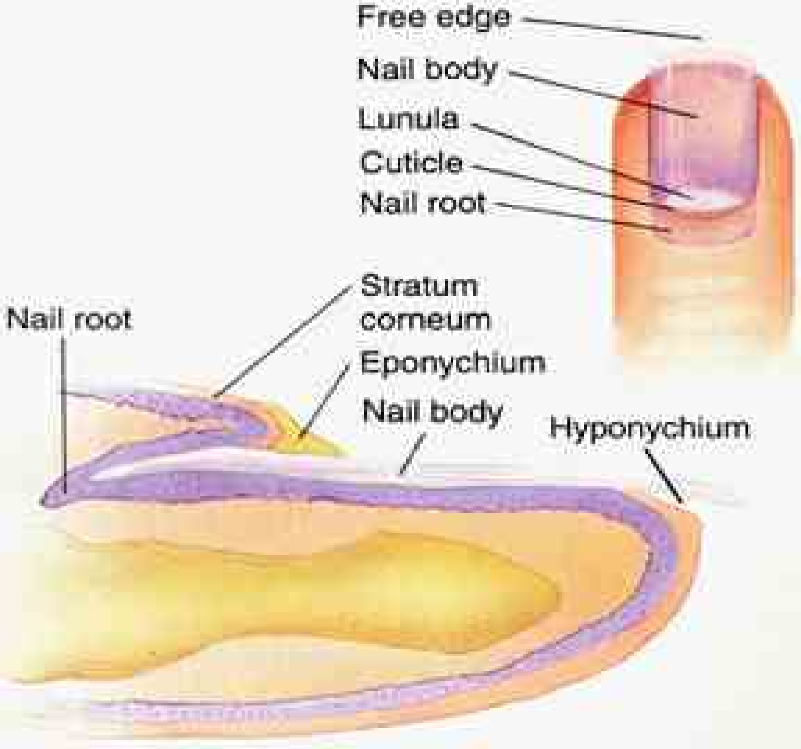
-**Apoptosis:** ‘Programmed cell death’ of individual cells.

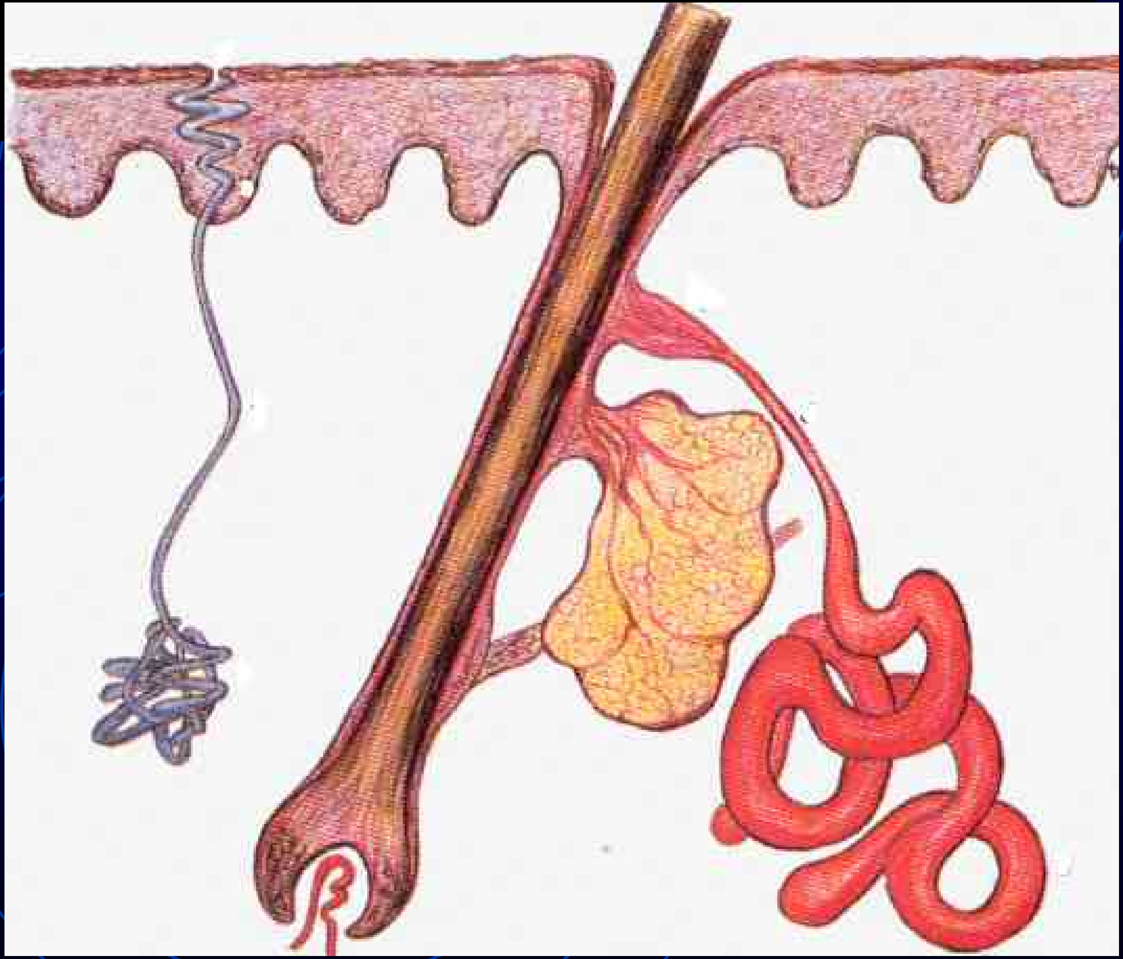
- **Atrophy:** Decreased thickness of the epidermis or dermis or both

-**Oedema:** Accumulation of interstitial fluid

Skin appendages:

- Hair
- Nails
- Sebaceous glands.
- Sweet glands.


Free edge
Nail body
Lunula
Cuticle
Nail root
Nail root
Stratum corneum
Eponychium
Nail body
Hyponychium



Functions of the skin:

The skin is the interface between the body and the external environment.

- **Protective:** It is the first line of defense against physical and thermal injury and infectious agents.
- **Prevention:** of water and electrolyte loss.
- **Temperature control**: is done by vasodilatation or constriction; and by vaporization of sweat from the skin surface.
- **mechanical barrier** against invading micro-organisms.
- **Sensory function** The nerves present in the skin are responsible for the different sensations such as temperature, pain, pressure and touch.
- **Formation of the vit D** from its precursor under the effect of sun light.
- **Immunological function** via langerhans cells.

Functions of normal skin

These include:

- i) Protective barrier against environmental insults
- ii) Temperature regulation
- iii) Sensation
- iv) Vitamin D synthesis
- v) Immunosurveillance
- vi) Appearance/cosmesis

Practical advice on topical treatments

- Emollients/moisturisers and complete emollient therapy Emollients are a key element in controlling and managing dry skin conditions. These products may be prescribed alone or be used as an adjuvant to other topical treatments such as topical steroids.
- **Causes of dry skin include:** environmental factors (dry air, exposure to wind); reduction in production of sebum (usually in old age); under active thyroid; inherited factors; and skin conditions (eczema, psoriasis).
- Complete emollient therapyThis is the term given to a regime which includes soap substitutes, bath oil and moisturiser.
- Soap substitutes – soap can have a drying effect on the skin so should be avoided in dry skin conditions. Soap substitutes, when applied prior to contact with water, help prevent stinging and act as a moisturiser.

- Bath additives – help to moisturise the skin while bathing. Some contain antipruritic and antistaphylococcal agents. These should only be used if indicated and for the prescribed period (usually 4–6 weeks). Patients should be advised not to purchase bath oils containing fragrance as it is a known sensitiser.
- *Advise the patient that bath additives can make the bath slippery.*
- Moisturisers Come as lotion, cream, gels, ointment and spray canisters.
- Lotions Are used for scalps or other hairy areas and for mild dryness on the face, trunk and limbs.
- CreamsCream -based products are the most commonly used moisturisers for dry skin conditions as they can be applied to the entire body, are cosmetically acceptable and have cooling properties.
- Gels Similar to creams.
- Ointments Are prescribed for drier, thicker, more scaly areas, but patients may find them too greasy.
- Spray canisters Quick to use.

- Application Patients should be advised to apply moisturiser directly to the skin in a downward motion in the direction of hair growth. This will reduce the risk of blocking the hair follicles (folliculitis).
- Moisturiser should be applied at least twice daily.
- It is important to prescribe the right amount of moisturiser. Prescribe 250g per week for a child under 10 years, and 600g for adults and children over 10 years.
- It is important to advise the patient, parent or carer not to stop complete emollient therapy once the condition is controlled as this will help to prevent future exacerbations.

Topical steroid therapy

- Topical steroids are extremely useful in inflammatory skin conditions such as eczema. In the UK, they are divided into four groups according to their potency.

Group	Strength
Weak	= to hydrocortisone
Moderately potent	2.5 times stronger
Potent	10 times stronger
Very potent	50 times stronger

- There are large numbers to choose from, but it is best to become familiar with a few, perhaps one or two from each group.
- The steroid ladder (opposite) identifies some of the steroids available and their potency. It also demonstrates the importance of titration of topical steroids: if the patient has been treated with a potent steroid and is improving, it is important to gradually reduce the potency, not discontinue treatment, to avoid a flare up of the condition.

- As a general rule, use the weakest possible steroid that is effective. For the face, 1% hydrocortisone or equivalent (weak) should be prescribed. Start with this elsewhere and only prescribe something stronger if this does not work. In general practice, it is rarely necessary to use anything stronger than a moderately potent steroid for extensive eczema. For localised persistent eczema, a potent steroid can be useful. Very potent steroids should rarely be used.
- 15g of ointment or cream is enough to cover the adult body once. To cover the whole body for a week if applied twice daily will require 200g. If a patient has widespread eczema, adequate amounts of topical steroid should be prescribed to avoid relapse. A useful guide to using the correct amount of topical steroid is the finger-tip unit guide, which is the amount of cream/ointment squeezed onto the index finger from tip to the first joint. This amount will cover the area of two palms.
- The potency of a steroid can be enhanced by the base used: for example, an ointment base makes a steroid more potent than a cream or lotion. Additionally, the ability of a topical steroid to be absorbed varies according to the site and the condition of the skin.
- It is important to prescribe complete emollient therapy along with topical steroid therapy as this will help to reduce inflammation and dryness.
- **Caution**
- Topical steroids are contraindicated in some skin conditions, including: acne

BACTERIAL INFECTIONS

Dr Abdelwassa Almekhlafi, MD

Definition:

- Pyogenic skin infection is a group of bacterial infections caused by strept or staph or both organisms.
- The surface of skin and hair follicles of normal healthy individuals are colonized many harmless microorganisms living as commensals which protect the skin from bacterial infections through bacterial interference.

The role of normal skin flora

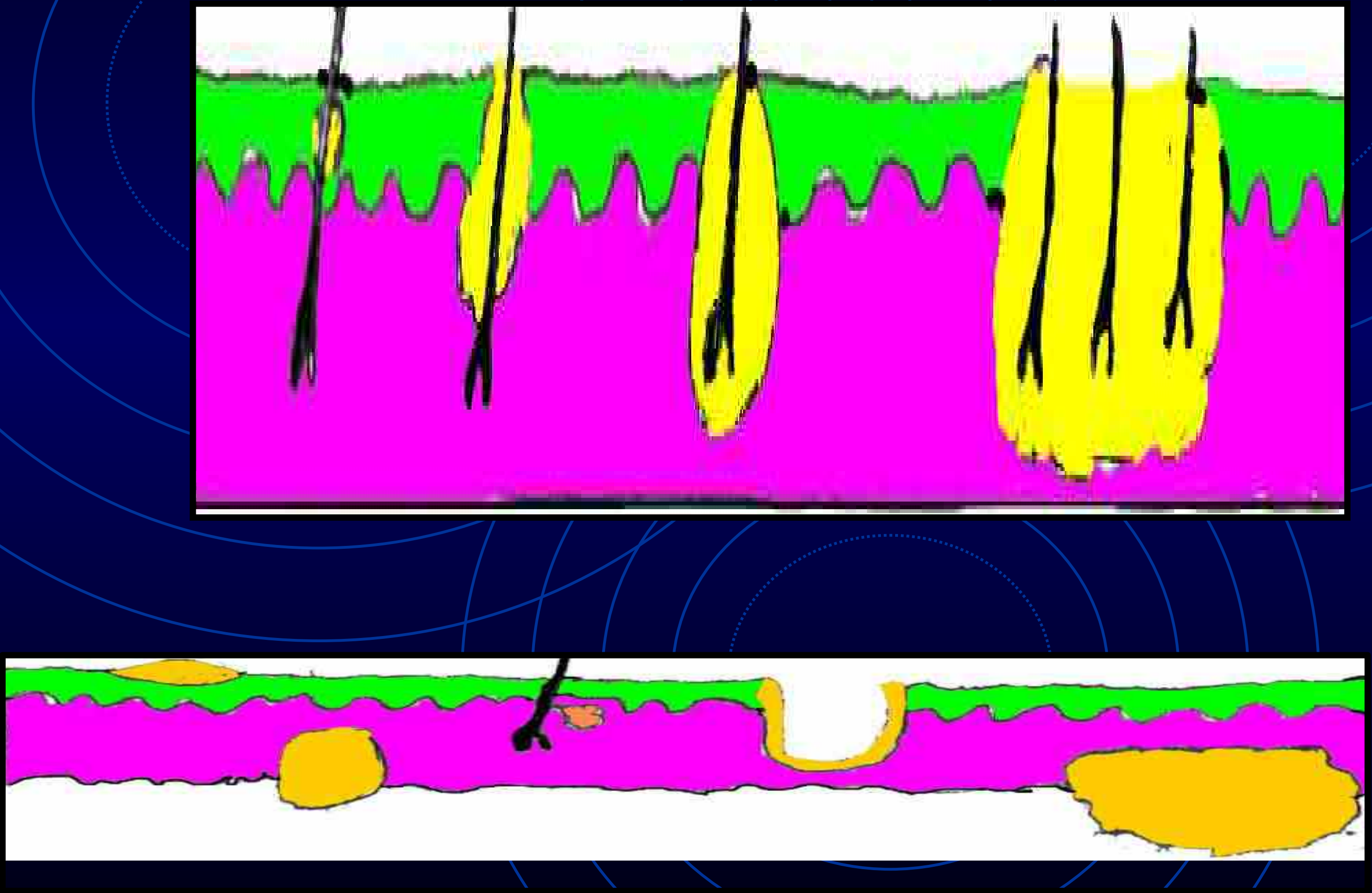
- - Defense against bacterial infection through bacterial interference (most important function)
- -Production of free fatty acids from skin lipids (P.acnes and G + cocci are capable of hydrolysing lipids of sebum to produce free fatty acids).
- -Apocrine sweet is sterile and odorless when secreted. The odor comes from bacterial action.

Skin defence against infection

- 1-acidic PH
- 2-relative dryness
- 3-desquamation
- 4-sweating
- 5-bacterial interference

streptococcal infections

- Impetigo
- Ecthyma
- Erysipelas
- Cellulitis.



Impetigo

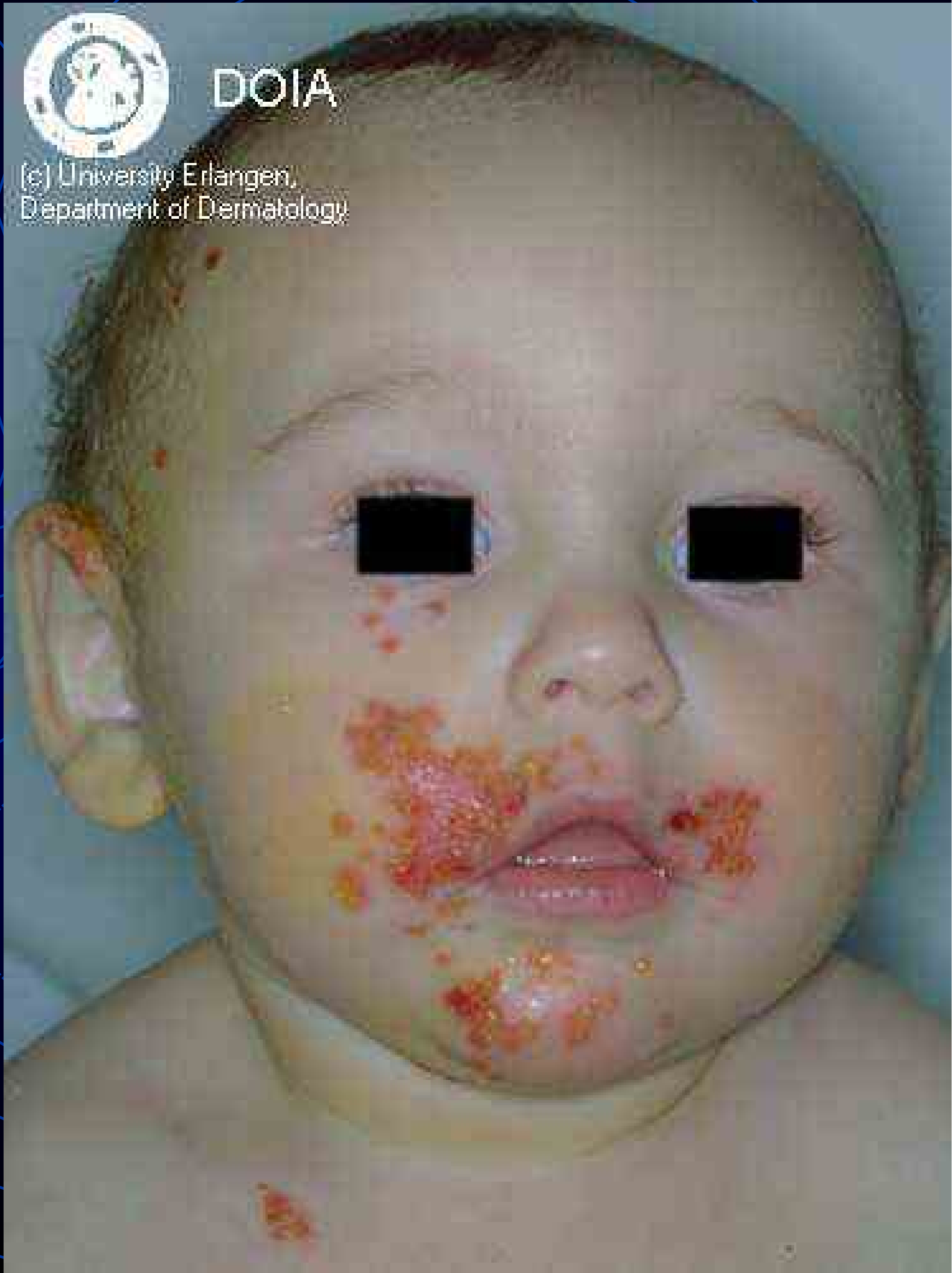
Impetigo is a contagious superficial pyogenic infection of the skin caused by staphylococci and streptococci.

It is more common in children and occurs mostly during summer.

Two main clinical forms are recognized:

- **Non-bullous impetigo**, may be caused by staphylococci, streptococci, or both organisms together.
- **Bullous impetigo**, usually caused by Staphylococcus aureus.

Predisposing factors: insect bites, pediculosis, scabies and minor trauma.

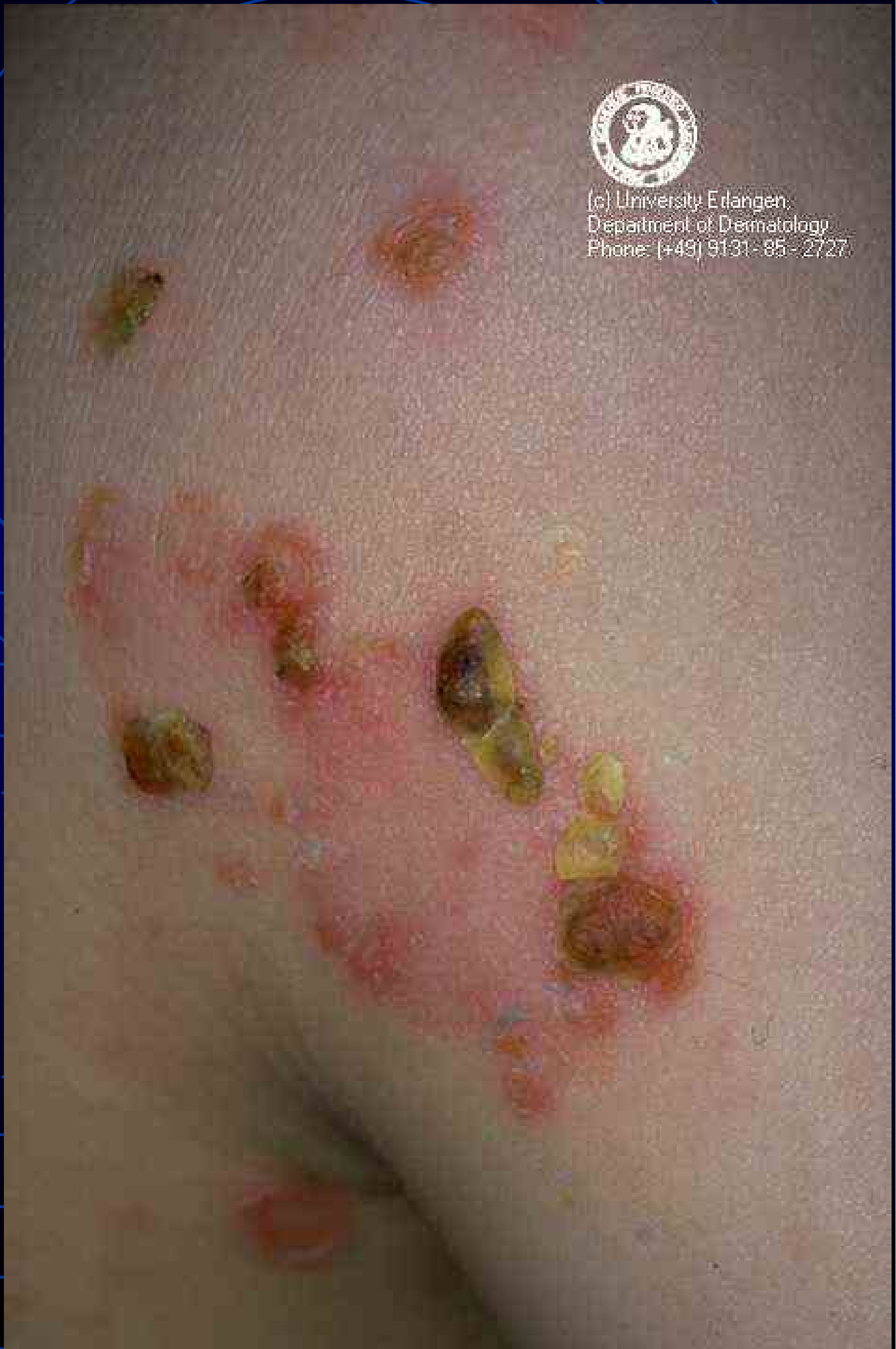

DOIA
(c) University Erlangen,
Department of Dermatology

Impetigo

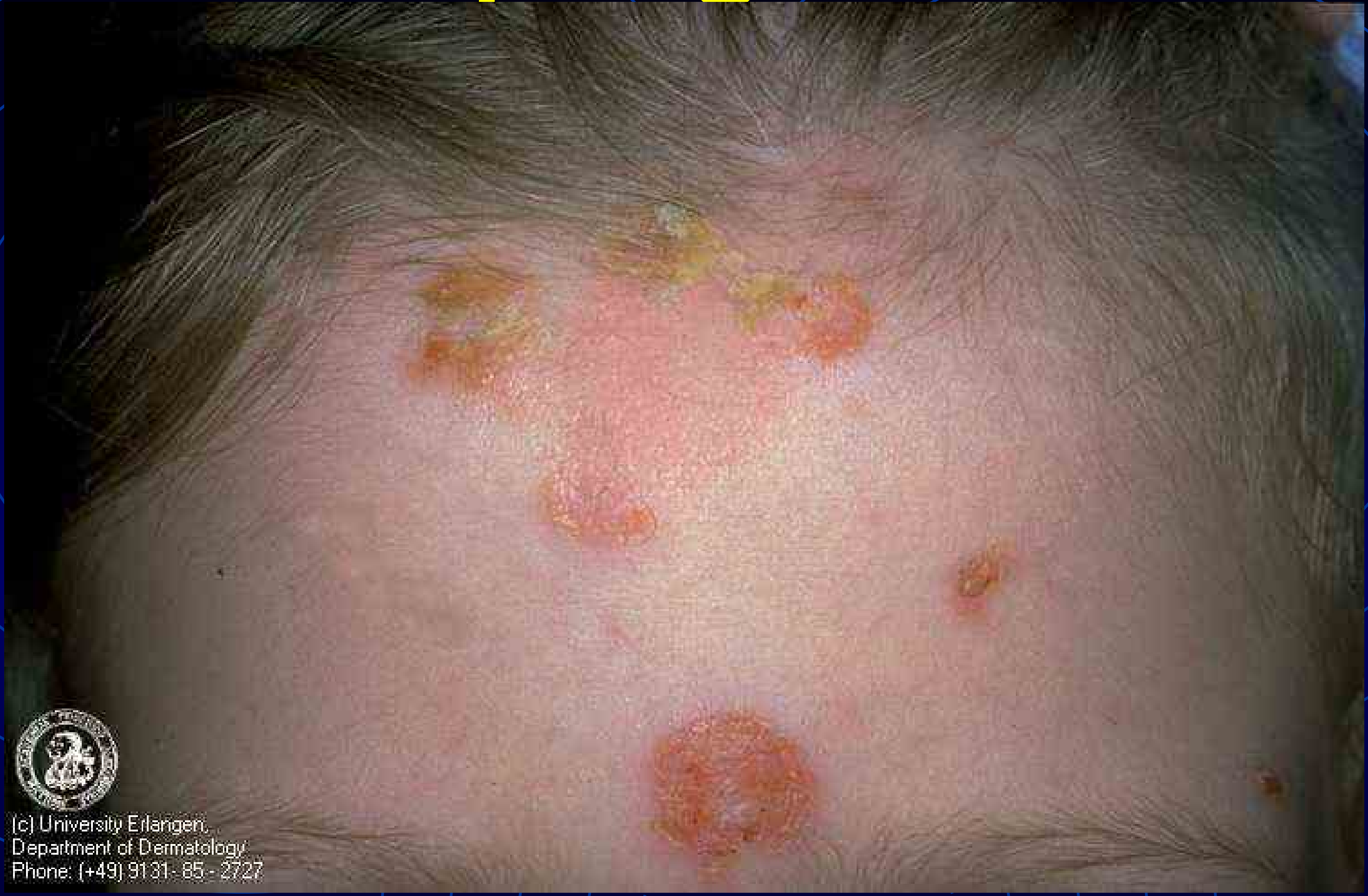
-Non-bullous impetigo

The initial lesion is a very thin walled vesicle, on an erythematous base. The vesicle ruptures so rapidly that it seldom seen as such. The exuding serum dries to form yellowish brown crust. Gradual peripheral extension occurs without central healing, and multiple lesions may coalesce. The crusts eventually dry and separate to leave erythema, which fades without scarring.

Spread of the lesions to other parts of the body occurs by fingers or towels.


(c) University Erlangen,
Department of Dermatology
Phone: (+49) 9131-85-2727

Impetigo



(c) University Erlangen,
Department of Dermatology
Phone: (+49) 9131- 85 - 2727

The face, especially around the nose and mouth, hands, neck and extremities are sites most commonly affected.

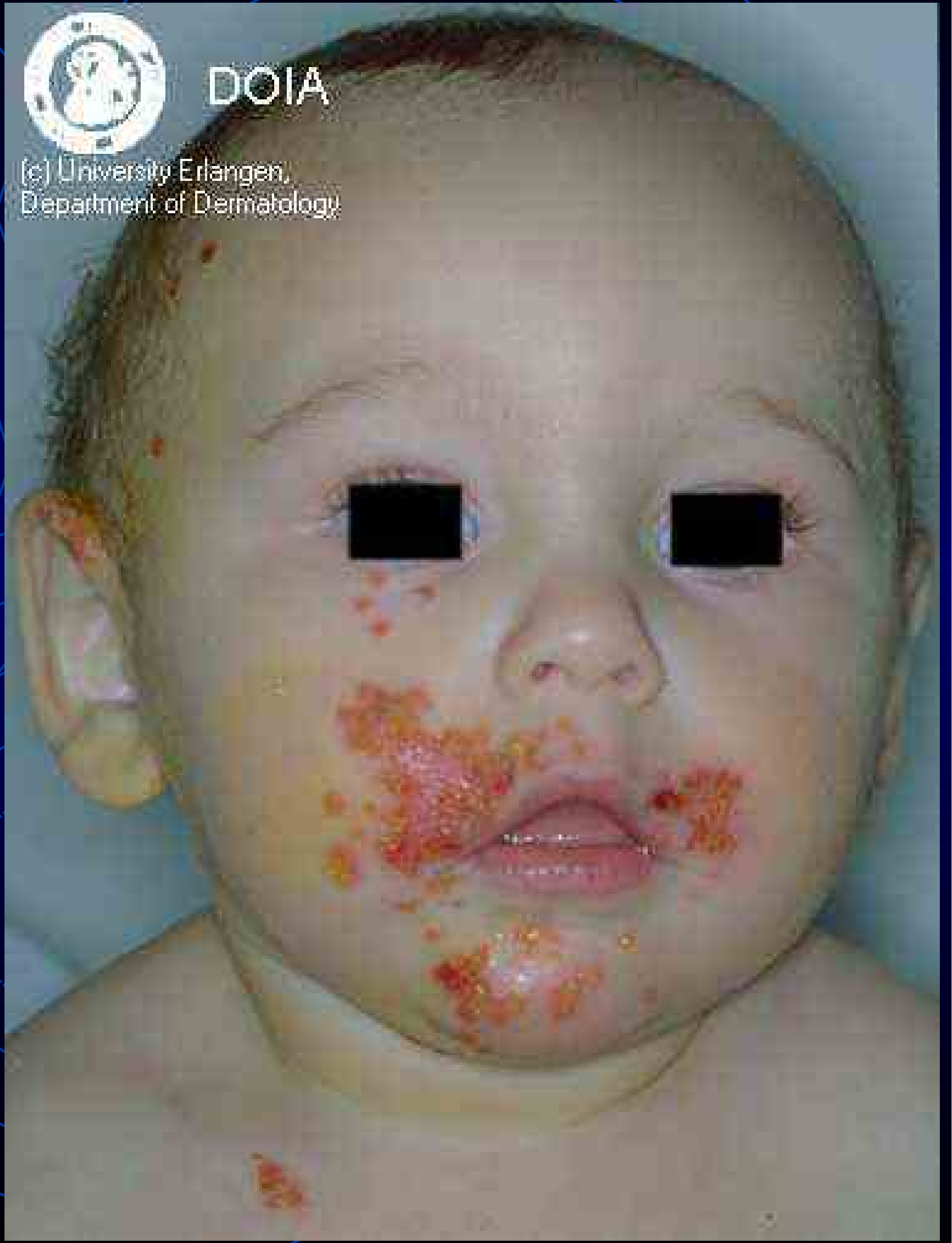
Involvement of the mucous membranes is rare.

Impetigo

Tendency to spontaneous cure occurs within 2-3 weeks but prolonged course is common.

In severe cases, there may be regional adenitis with fever and other constitutional symptoms.

Impetigo of the scalp is a frequent complication of pediculosis capitis.



(c) University Erlangen, Department of Dermatology

-Bullous impetigo

The bullae are few in number, less rapidly ruptured, much larger and may persist for 2 to 3 days occur anywhere on the body.

Contents are first clear then cloudy then have the same course as non bullous impetigo.

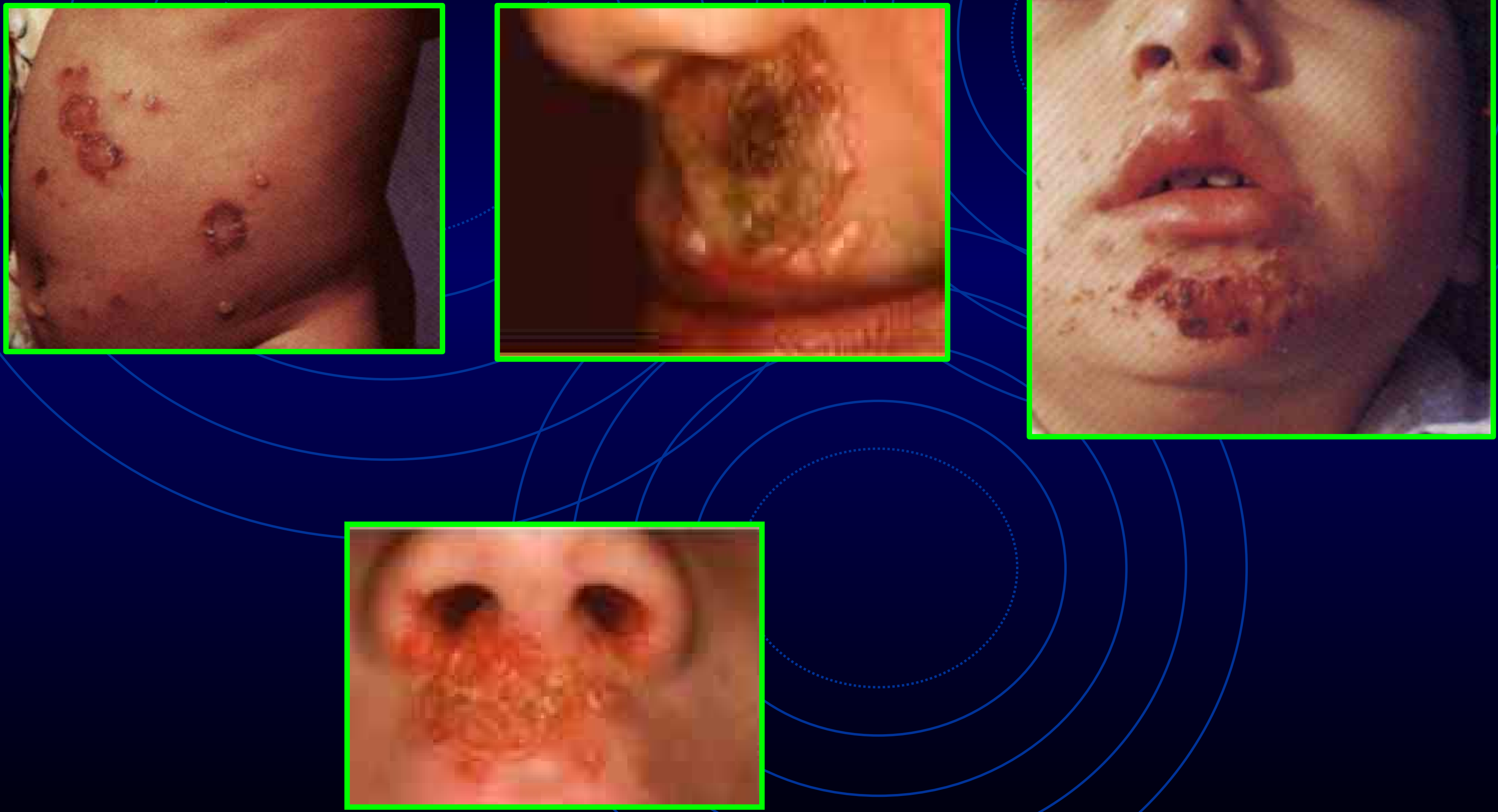
-*Circinate impetigo*

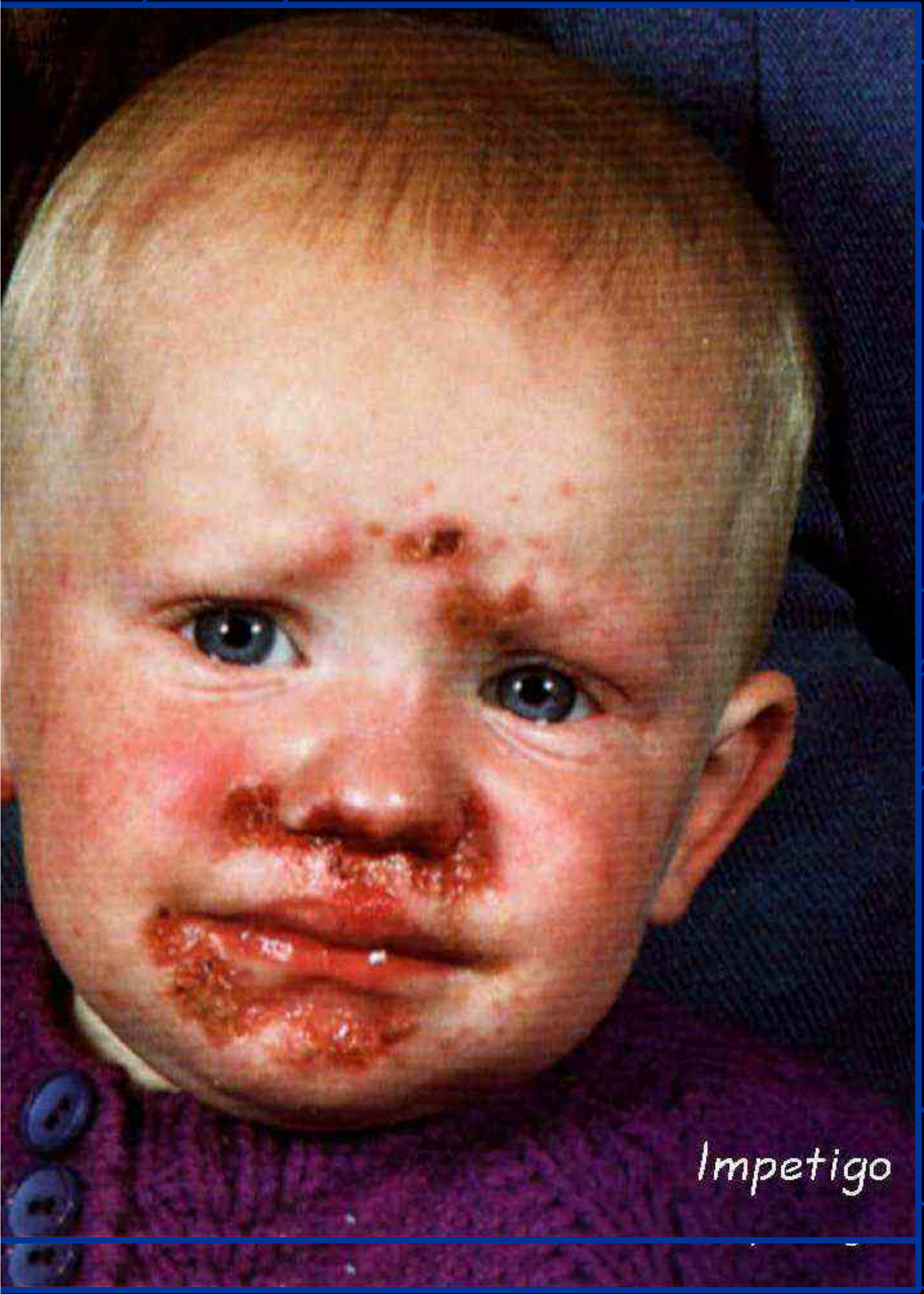
Complications

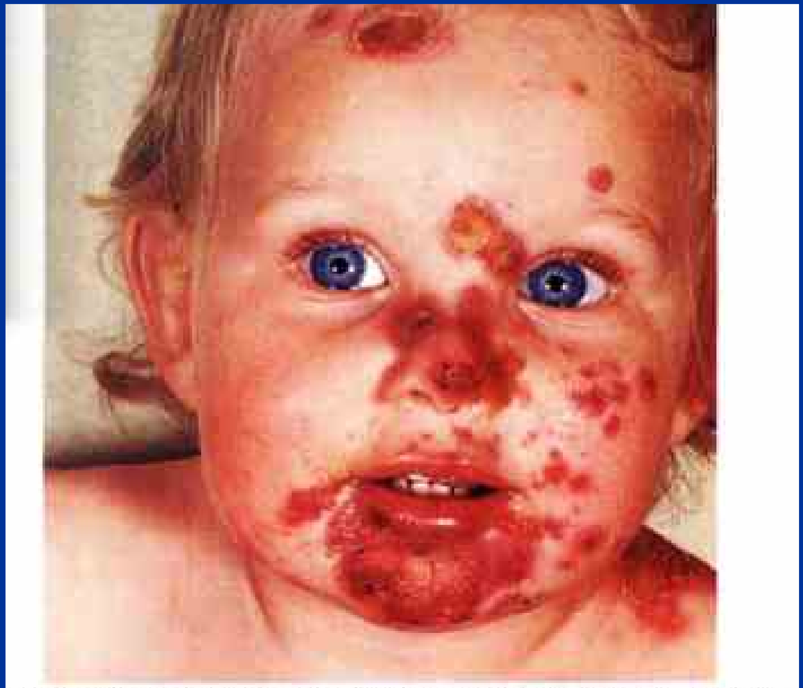
-**Acute glomerulonephritis** may occur as a complication of group A beta-hemolytic streptococcal skin infection.

-**Spread of infection** to the same patient and to others,

-**Rheumatic fever** is not a complication of streptococcal impetigo







Impetigo. *Extensive involvement of the face with scattered, discrete lesions and large confluent lesions; there are golden-yellow "stuck-on" crusts.*

Treatment

Control of predisposing factors e.g. insect bites, pediculosis, scabies and minor trauma.

Topical antibiotics such as fusidic acid and bacitracin are used alone in mild and localized infections.

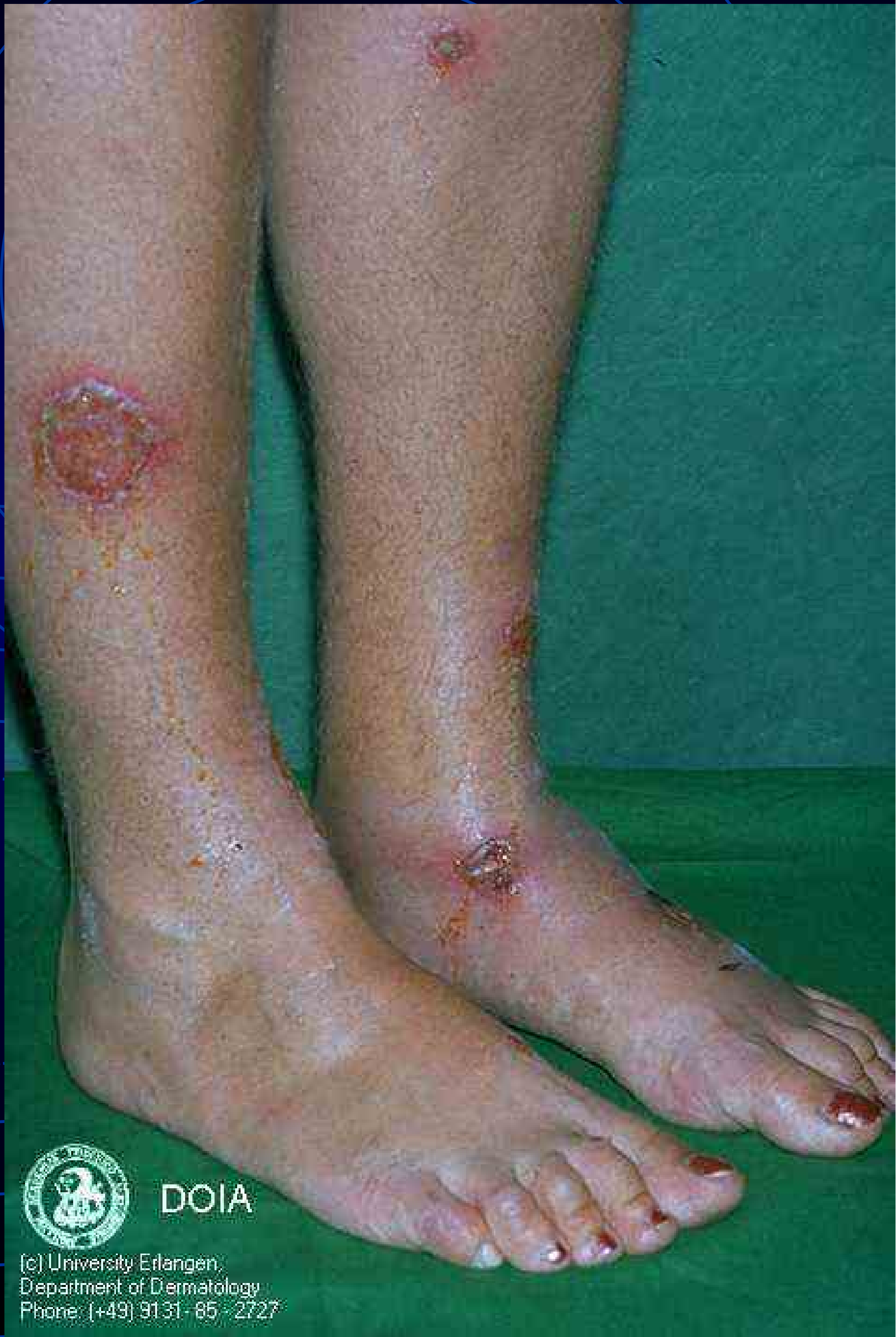
Systemic antibiotics such as flucloxacillin or erythromycin are indicated in widespread or severe infections.

Ecthyma

IT is a pyogenic infection of the skin caused by staphylococci and streptococci.

It is characterized by the formation of adherent crusts beneath which ulceration occurs.

In Europe, most cases occur in children, but in tropics it may occur at any age.

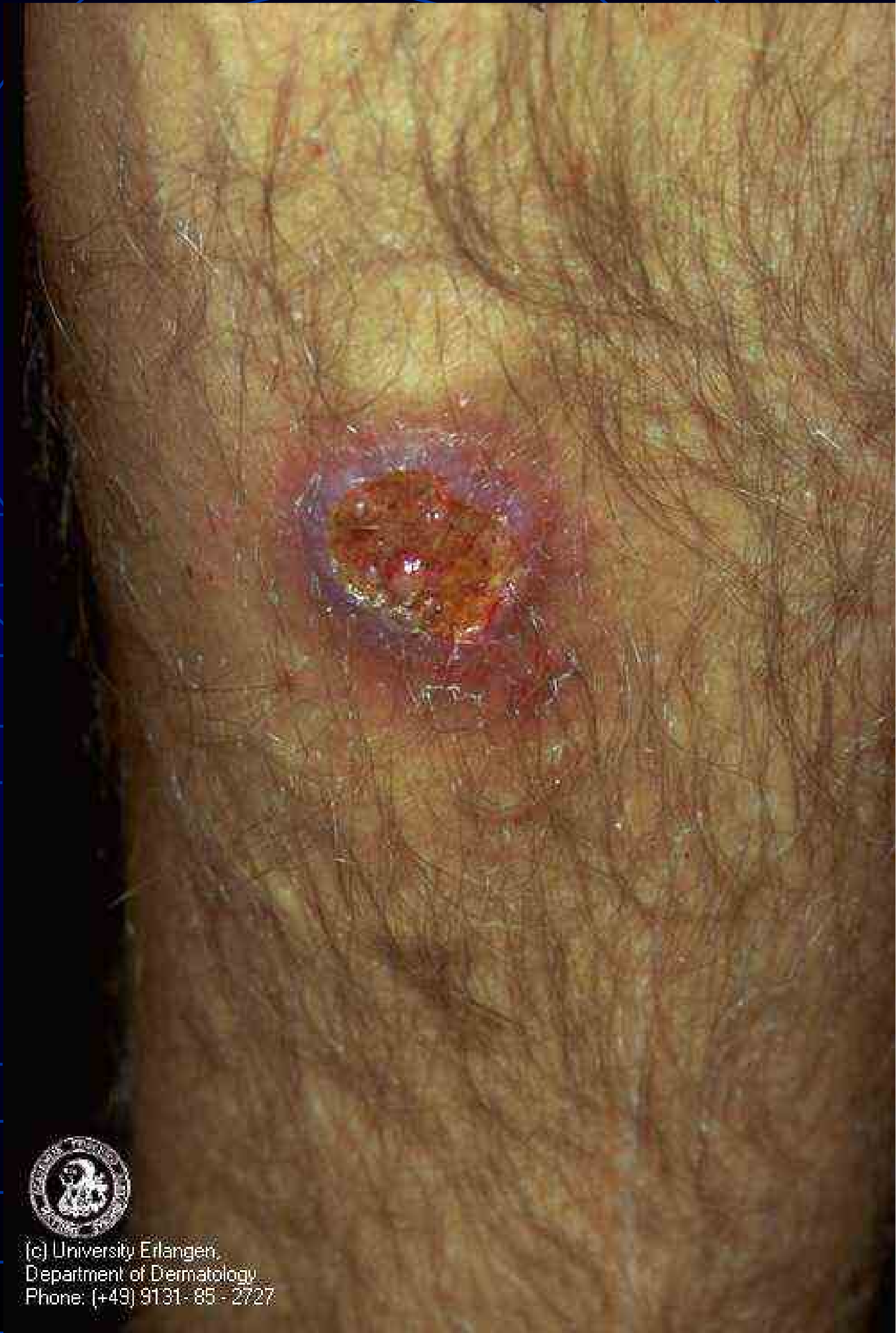


DOIA

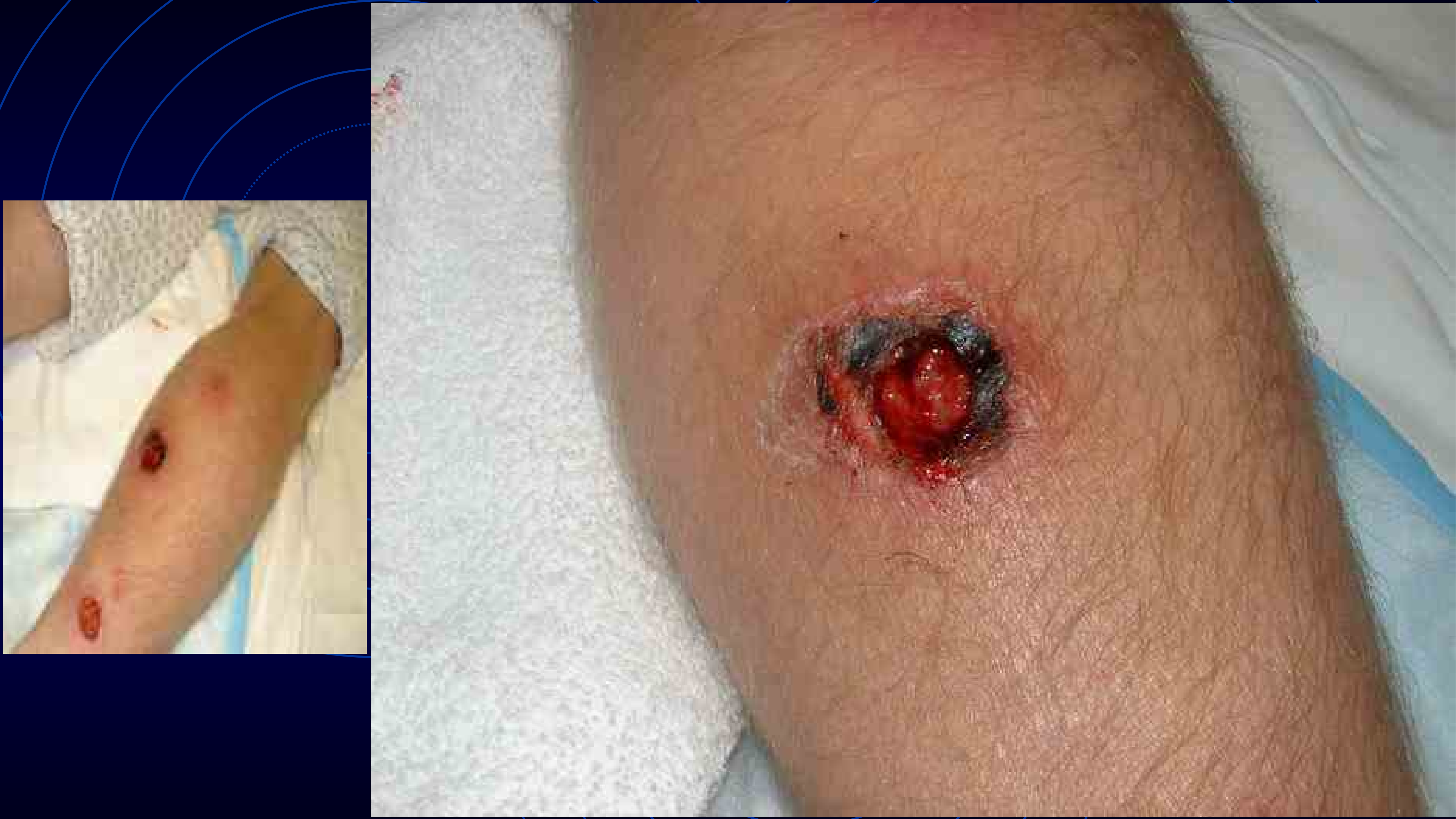
(c) University Erlangen,
Department of Dermatology
Phone: (+49) 9131 - 85 - 2727

Ecthyma

- Clinically small bullae or pustules on an erythematous base is soon surmounted by a hard crust of dried exudate which increases in size by peripheral rim accretion.
- The crust is removed with difficulty to reveal a purulent irregular ulcer.
- Healing occurs after few weeks with scarring.



(c) University Erlangen,
Department of Dermatology
Phone: (+49) 9131-85-2727

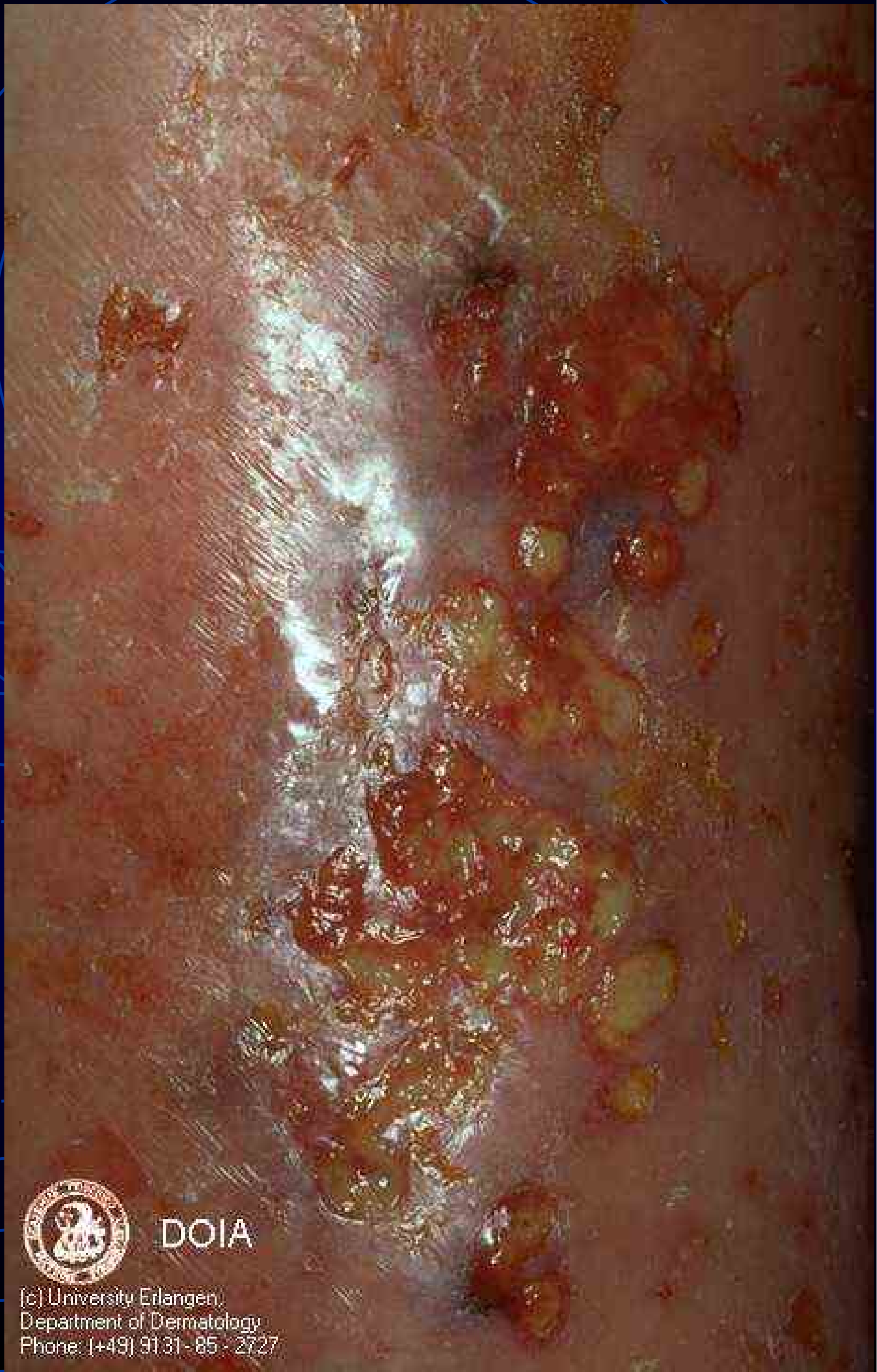


Ecthyma

The lesions are usually few in number and the buttocks, thighs, and legs are most commonly affected.

Treatment

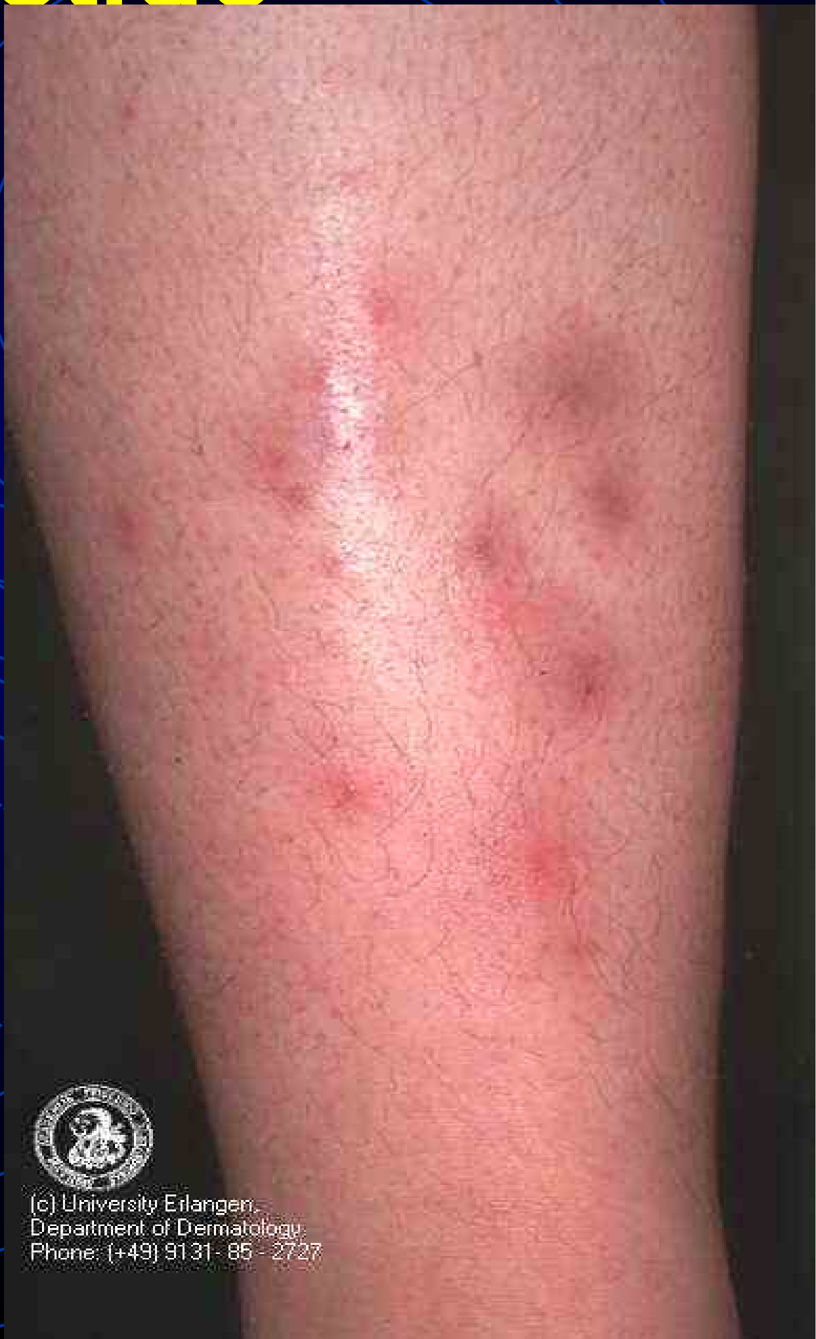
Topical and systemic treatment as for impetigo.



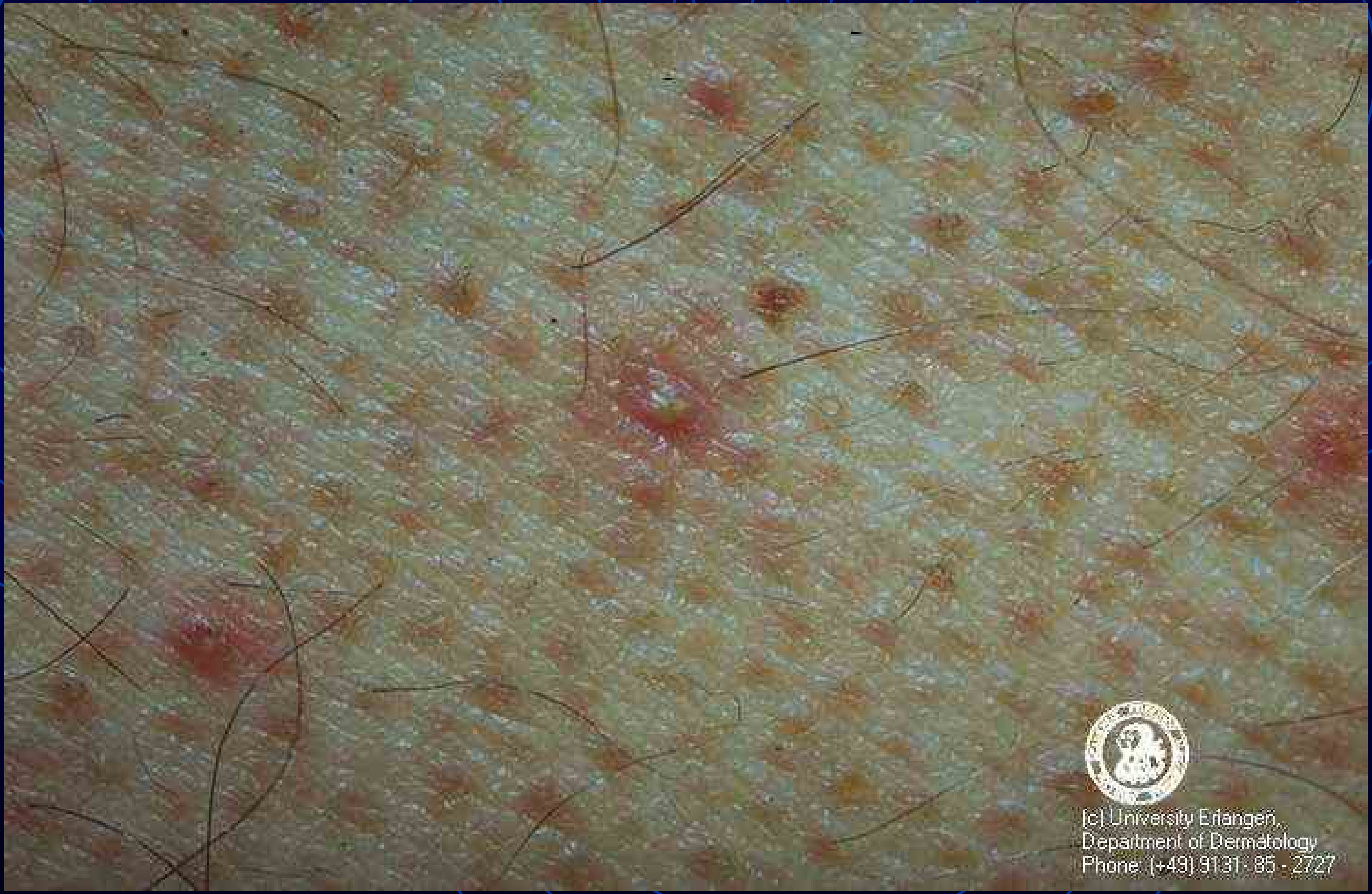
(c) University Erlangen,
Department of Dermatology
Phone: (+49) 9131-85-2727

Folliculitis
Bockhart's impetigo

- *Superficial folliculitis (Bockhart's impetigo)*
- It is an infection of the follicular orifices with *Staphylococcus aureus*. It appears as fragile yellow pustules and affects mainly the extremities and scalp.



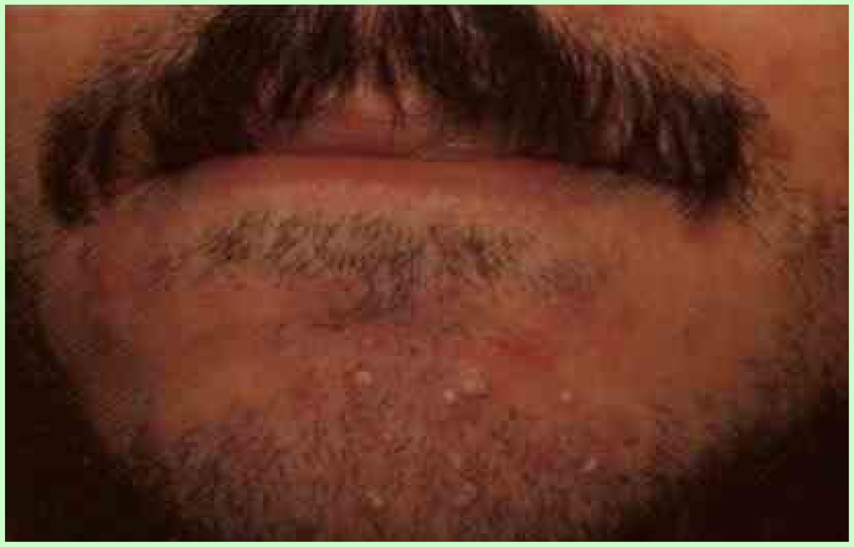
(c) University Erlangen,
Department of Dermatology
Phone: (+49) 9131- 85 - 2727

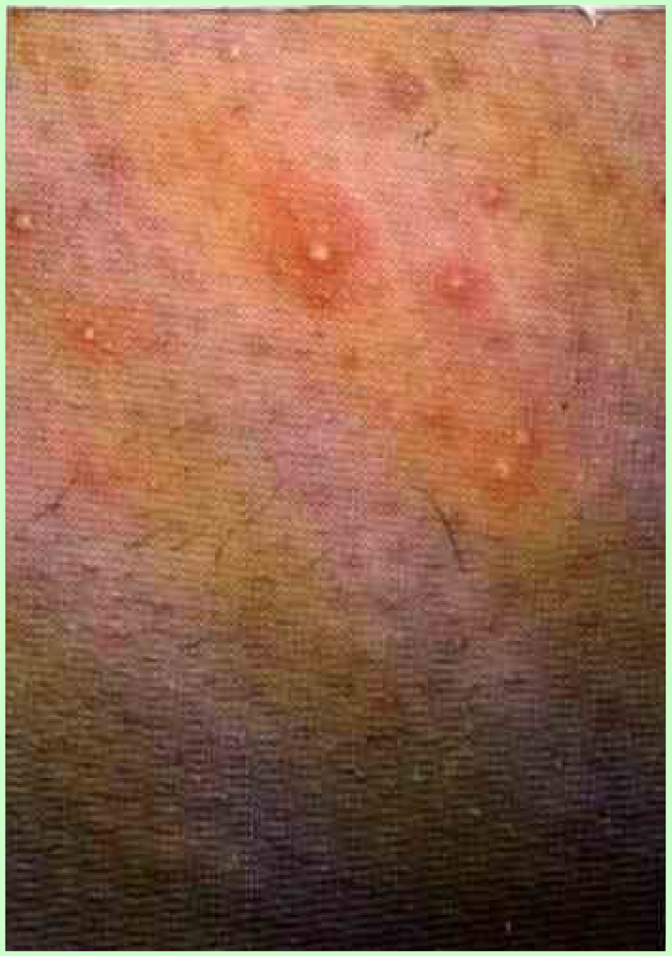


(c) University Erlangen,
Department of Dermatology
Phone: (+49) 9131- 85 - 2727

Folliculitis
Sycosis vulgaris

- It is a perifollicular, chronic, pustular staphylococcal infection of the beard region, characterized by inflammatory papules and pustules, and a tendency to recurrence.





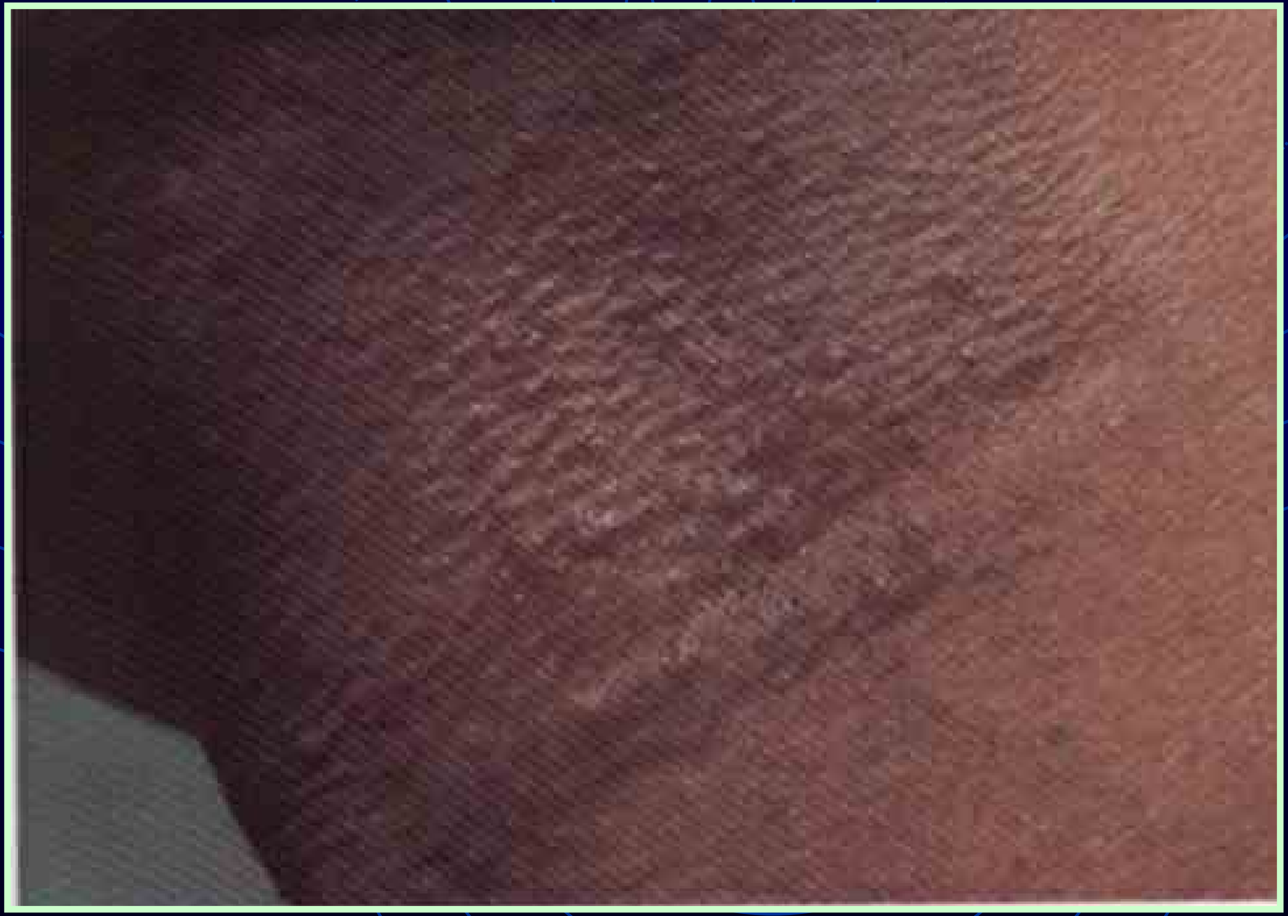
Folliculitis

pseudofolliculitis

Pseudofolliculitis barbae: caused by ingrowing hairs where the papules and pustules are present in the sides and angles of the jaw.

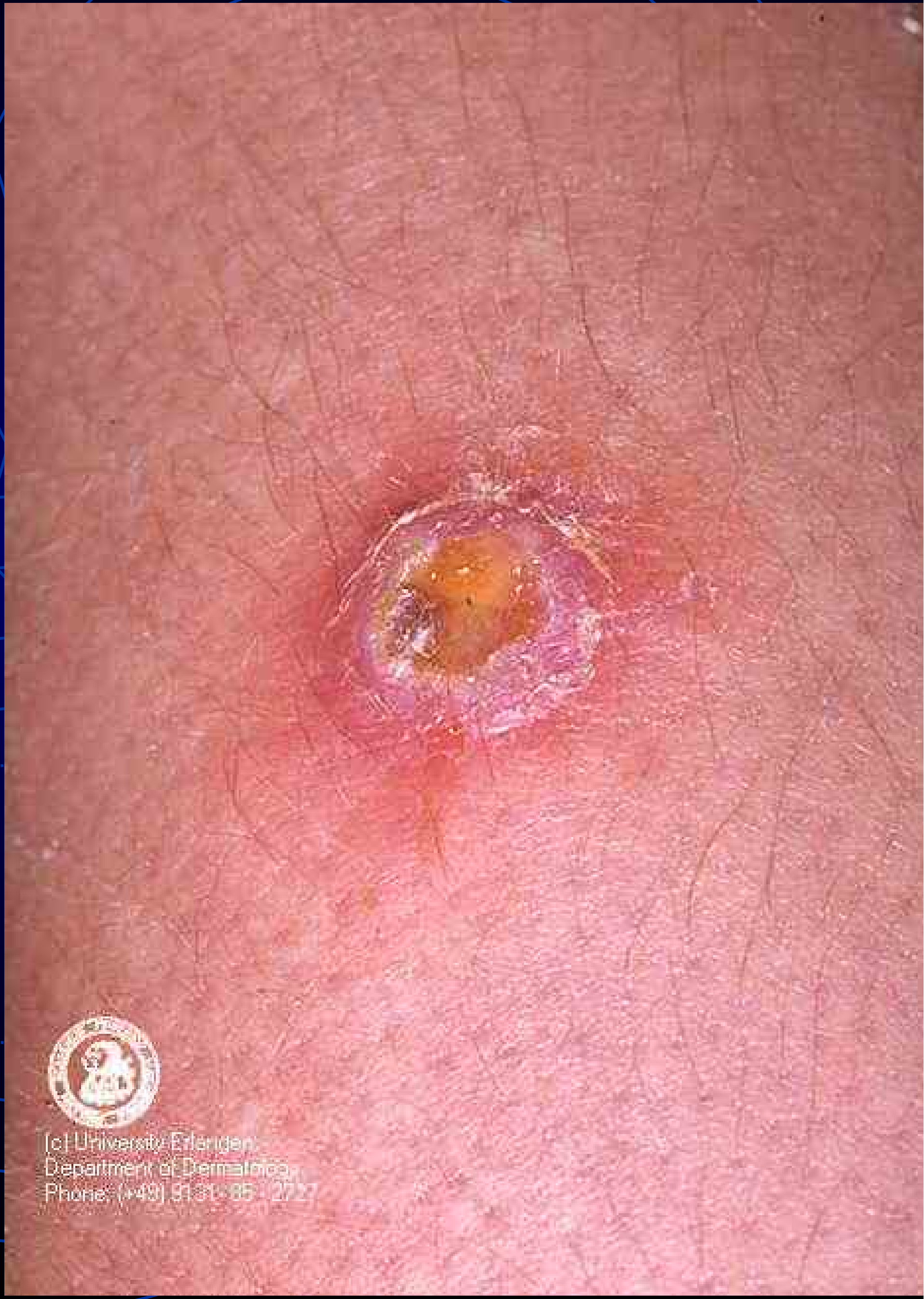
Treatment:

- Treatment of the predisposing factors.
- Topical antiseptics.
- Topical and systemic antibiotics.



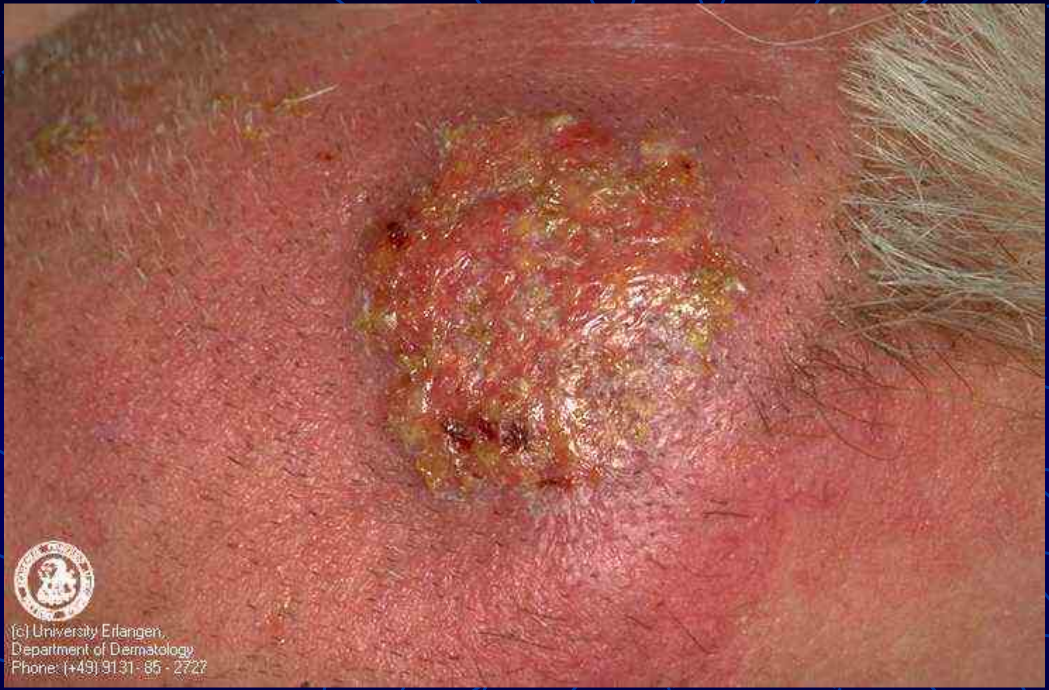
Furunculosis (Boils)

A furuncle, or boil, is an acute, round, tender, circumscribed, follicular staphylococcal abscess that generally ends in central suppuration.



(c) University Erlangen
Department of Dermatology
Phone: (+49) 9131-85-2727

Carbuncle



(c) University Erlangen,
Department of Dermatology
Phone: (+49) 9131 - 85 - 2727

A carbuncle is merely two or more confluent furuncles, with separate heads.

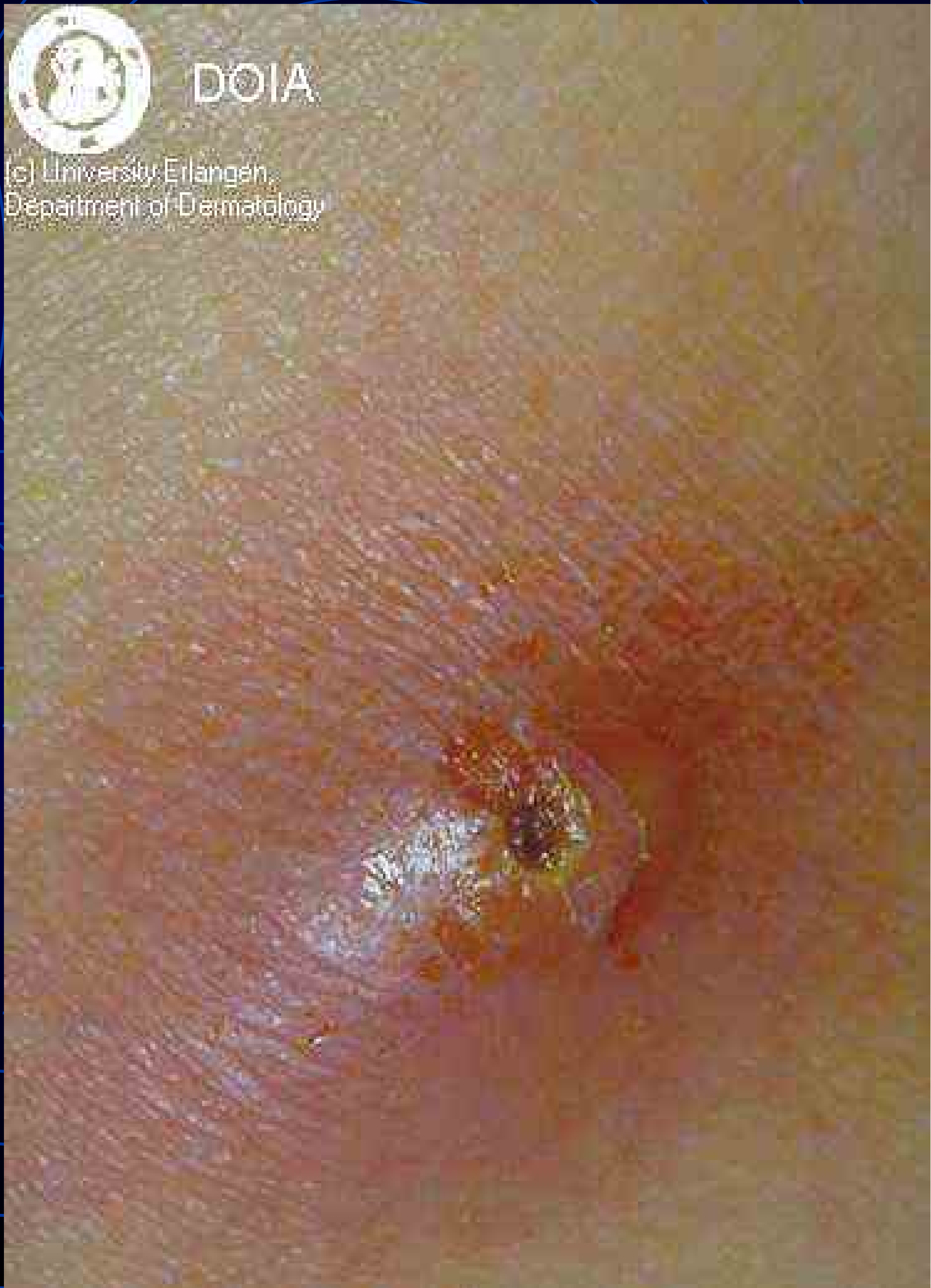
Furunculosis (Boils)

- Predisposintg facors

Impaired integrity of the skin surface by irritation, friction, hyperhidrosis, dermatitis or shaving.

Presence of a contagion, or autoinoculation from a carrier focus, usually in the nose or groin.

Systemic disorders as malnutrition, blood diseases, diabetes, and immunosuppressive conditions as AIDS.

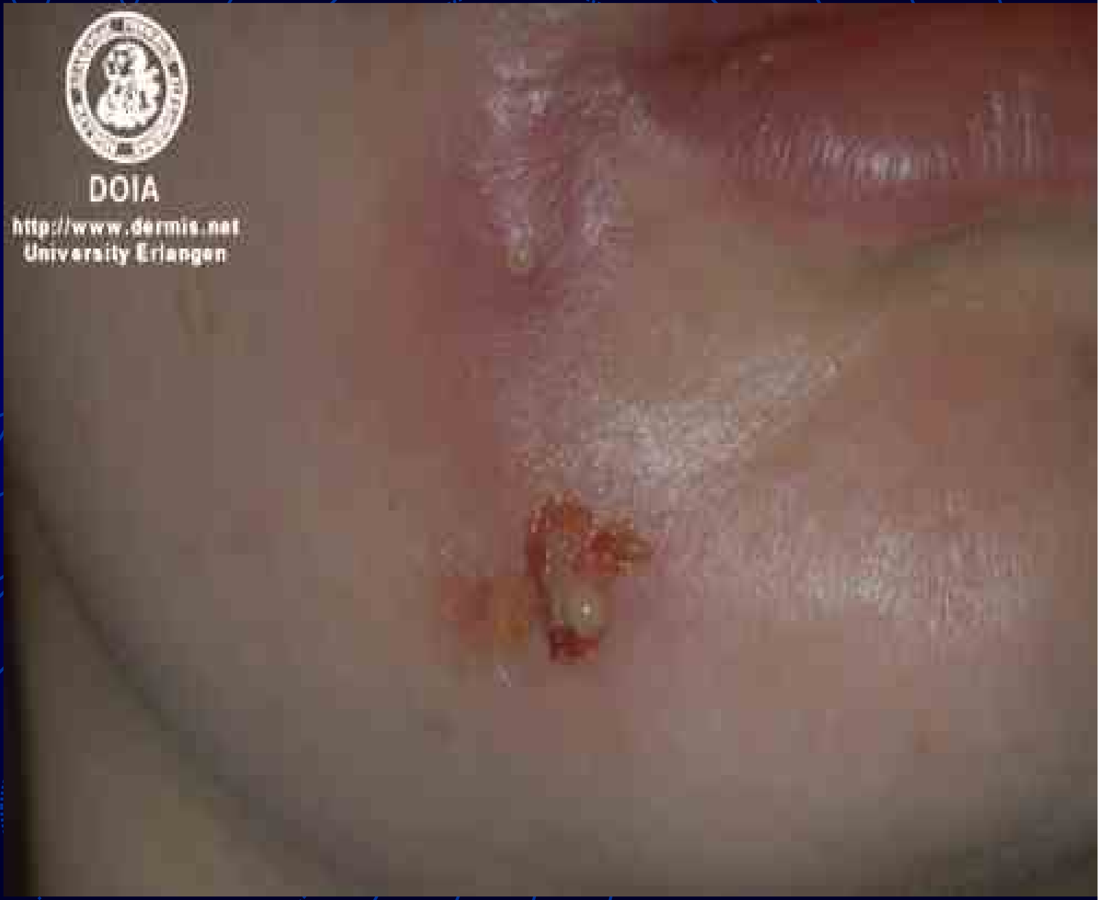

DOIA
(c) University Erlangen,
Department of Dermatology

Furunculosis (Boils)

The lesions begin in hair follicles, and often continue for a prolonged period by autoinoculation.

Some lesions disappear before rupture, but most undergo central necrosis and rupture through the skin, discharging purulent, necrotic debris.

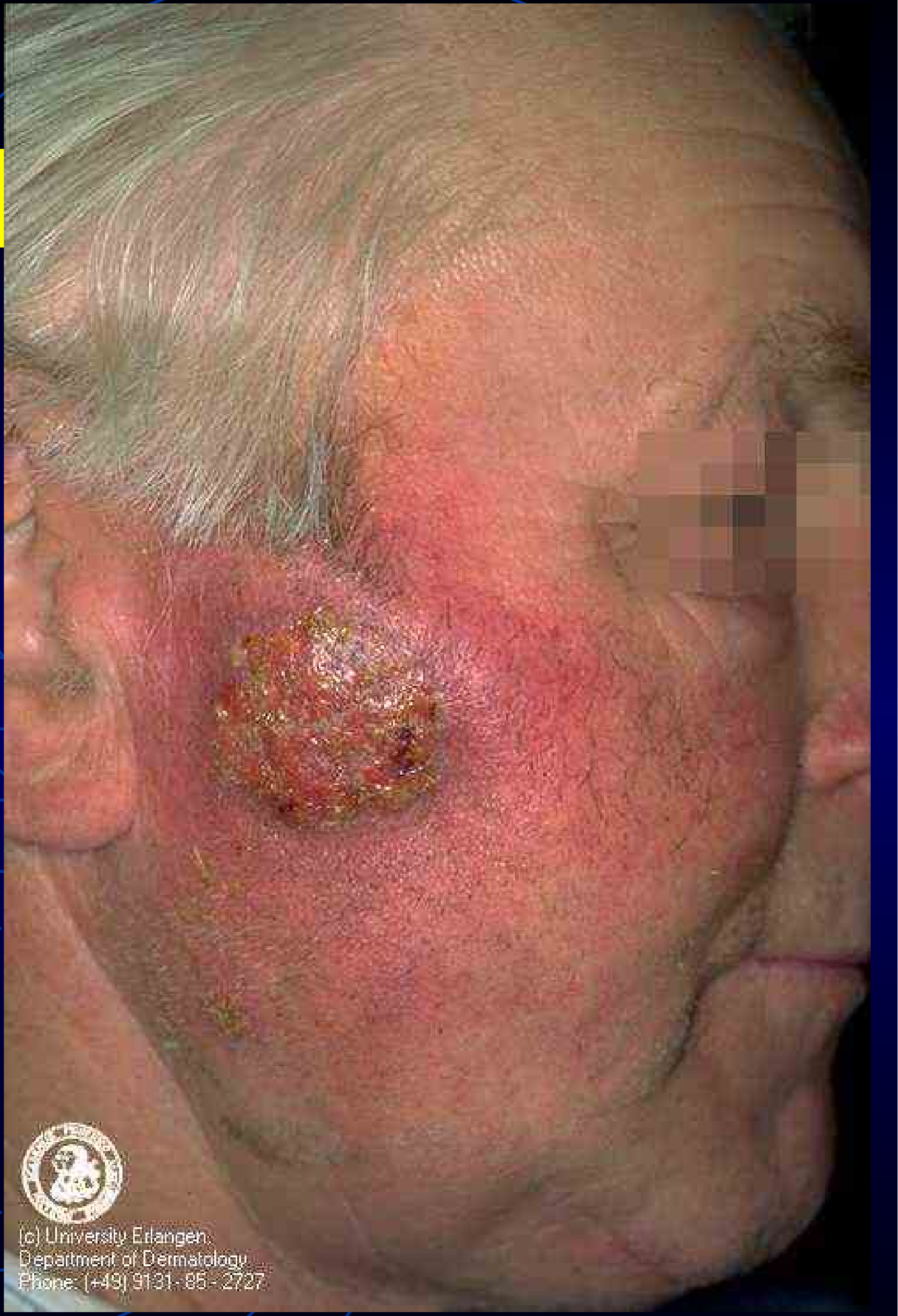
The sites commonly involved are the face and neck, the arms, wrists and fingers, the buttocks and the anogenital region.


DOIA
http://www.dermis.net
University Erlangen

Furunculosis (Boil

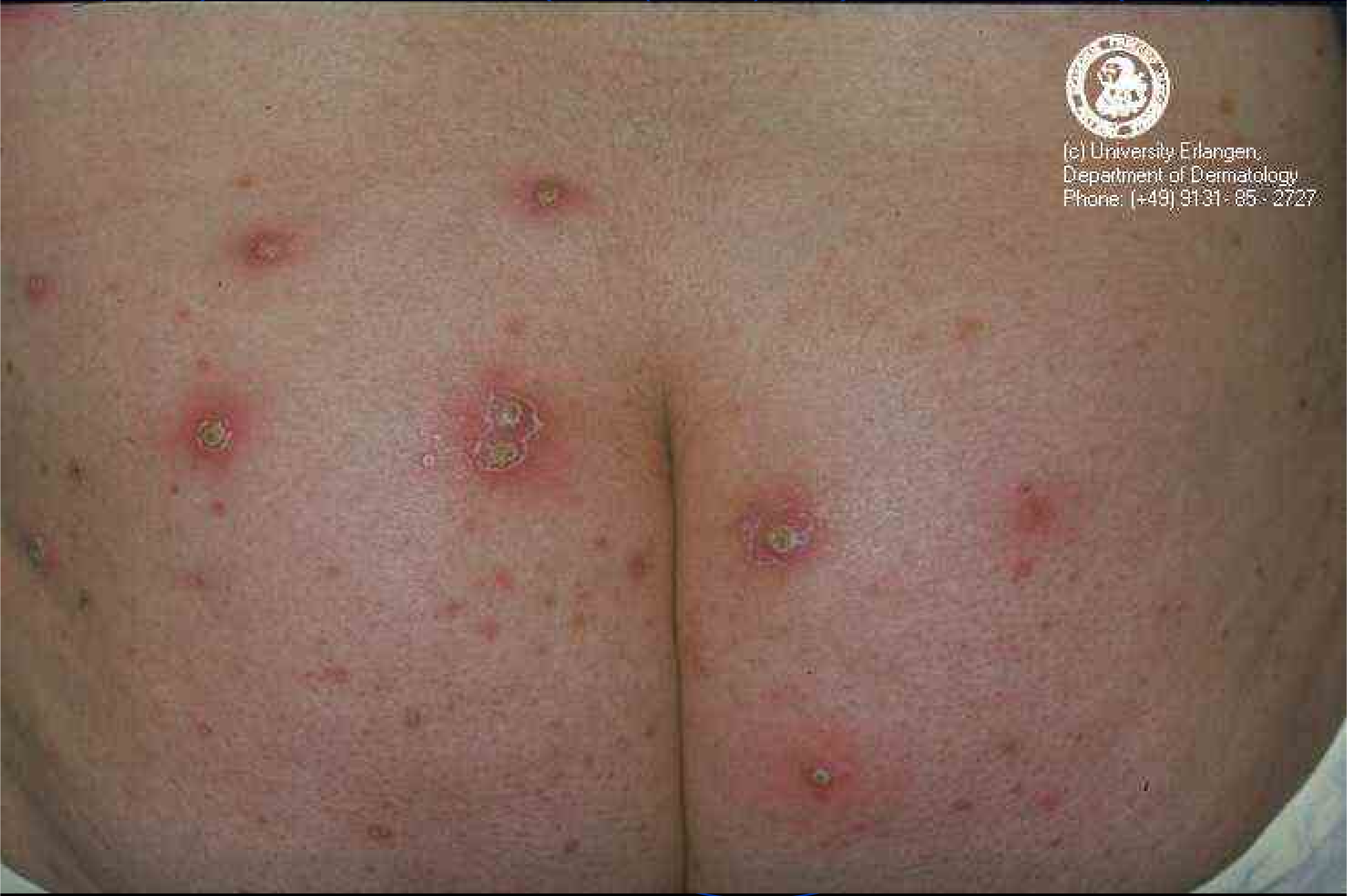
Treatment

- Topical and systemic antibiotics as for impetigo.
- Incision and drainage of some cases.

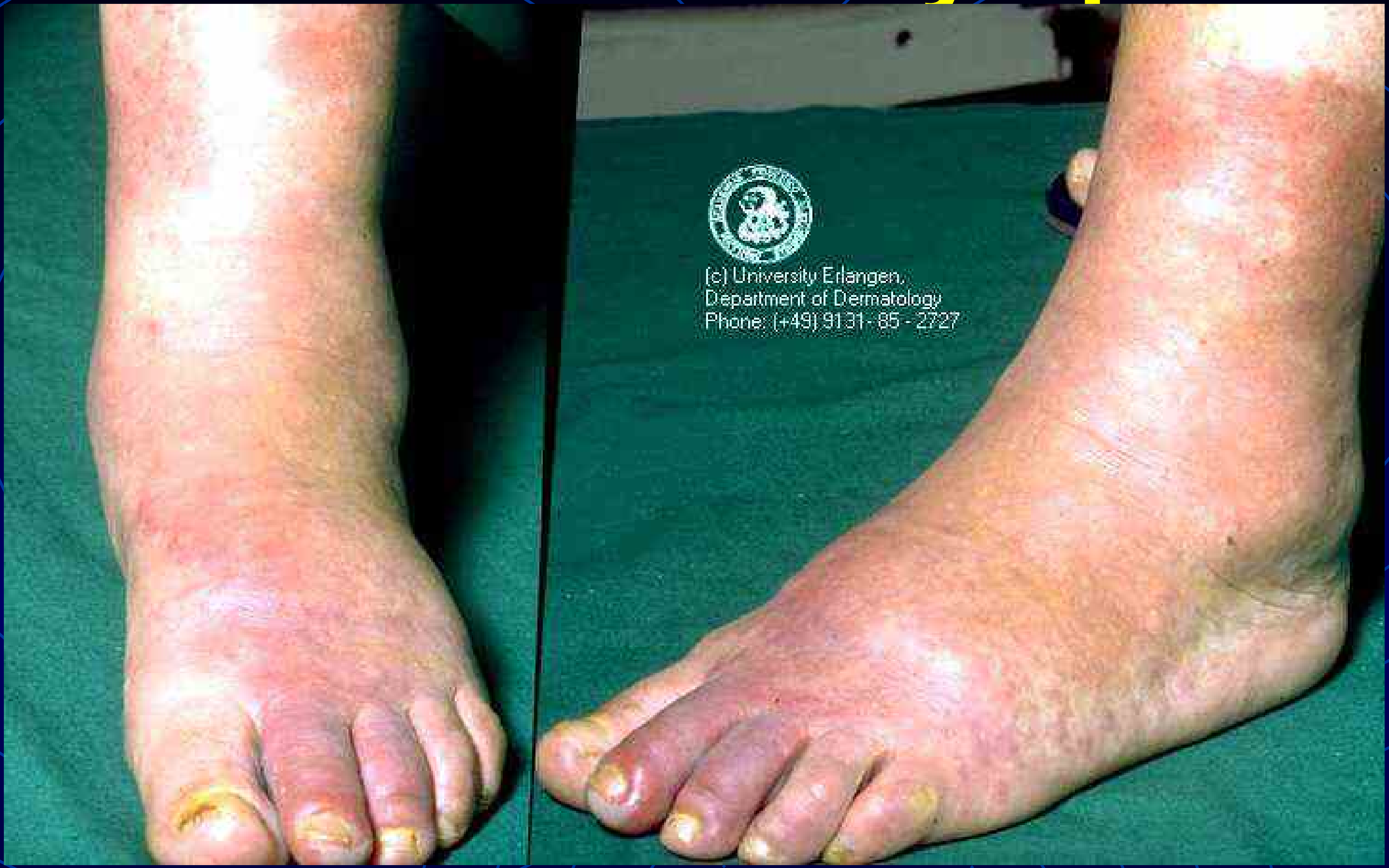


(c) University Erlangen,
Department of Dermatology
Phone: (+49) 9131-85-2727

What is your diagnosis


(c) University Erlangen,
Department of Dermatology
Phone: (+49) 9131-85-2727

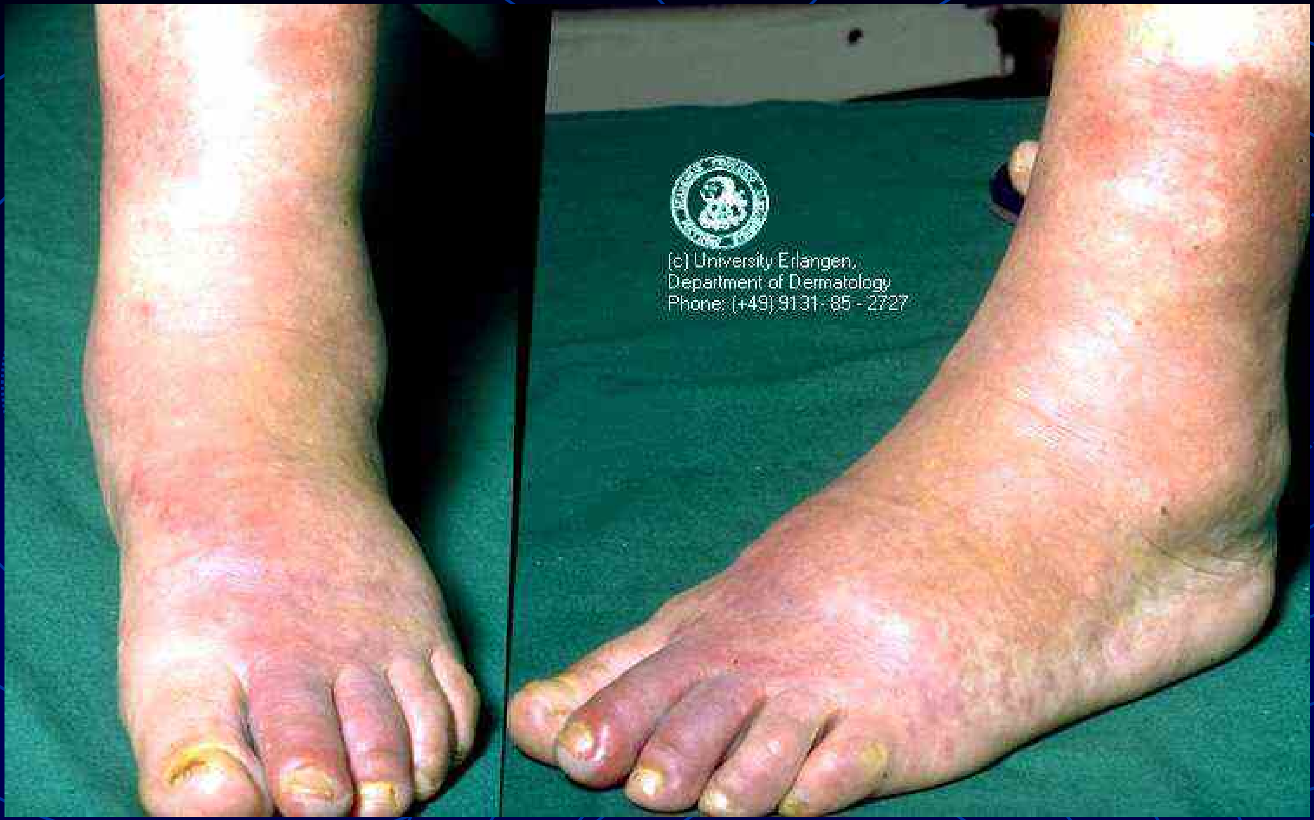
Cellulitis and Erysipelas



(c) University Erlangen, Department of Dermatology Phone: (+49) 9131 - 85 - 2727

Cellulitis is an infection of the subcutaneous tissue, while erysipelas is more superficial as it involves the dermis and upper subcutaneous tissue. However, cellulitis may extend superficially and erysipelas deeply so that the two conditions overlap.

Cellulitis and Erysipelas



(c) University Erlangen,
Department of Dermatology
Phone: (+49) 9131-85-2727

- Current view tends to regard erysipelas as a form of cellulitis rather than a distinct entity. Cellulitis and erysipelas are caused mainly by *Streptococcus pyogenes*

Clinical picture

- Erythema, heat, swelling and pain or tenderness are constant features.

In erysipelas, the edge of the lesion is well-demarcated and raised, but in cellulitis it is diffuse.

Also, in erysipelas blistering and hemorrhage are more common. Lymphangitis and lymphadenopathy are frequent.

Except in mild cases, there is constitutional upset with fever and malaise, more severe in erysipelas.

The face and the legs are the most frequent sites affected.

Predisposing causes are wounds, fissures, ulcers or inflammatory lesions such as interdigital fungal or bacterial infection.

Of the unusual complications are gangrene, metastatic abscesses and grave sepsis.

Treatment

Systemic penicillin and cephalosporins are usually effective.

Erysipelas and Cellulitis

Description *Spreading bacterial infection of the skin*

Cellulitis **A purulent inflammation of the skin and subcutaneous tissue**

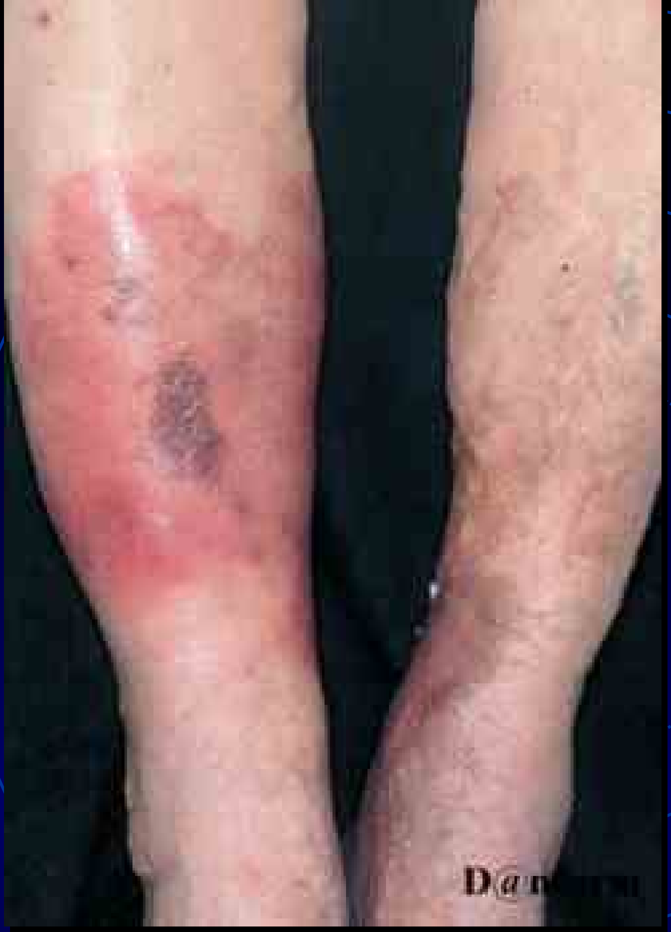
Erysipelas **is an acute superficial form of cellulitis and involves** the dermis and upper subcutaneous tissue

- *Causes Streptococcus pyogenes*
- Risk factors include immunosuppression, wounds, leg ulcers, toeweb intertrigo, and minor skin injury
- *Presentation Most common in the lower limbs*

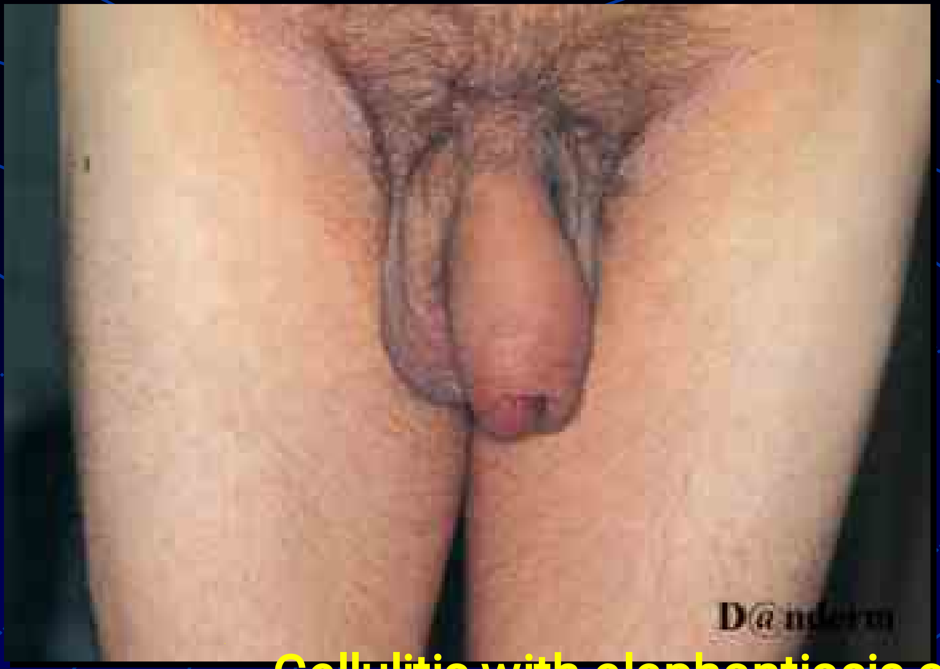
  Local signs of inflammation – swelling (tumor), erythema (rubor), warmth (calor), pain (dolor); may be associated with lymphangitis

  Systemically unwell with fever, malaise or rigors, particularly with erysipelas
- **Erysipelas is distinguished from cellulitis by a well-defined, red** raised border
- *Management Antibiotics (e.g. flucloxacillin or benzylpenicillin)*

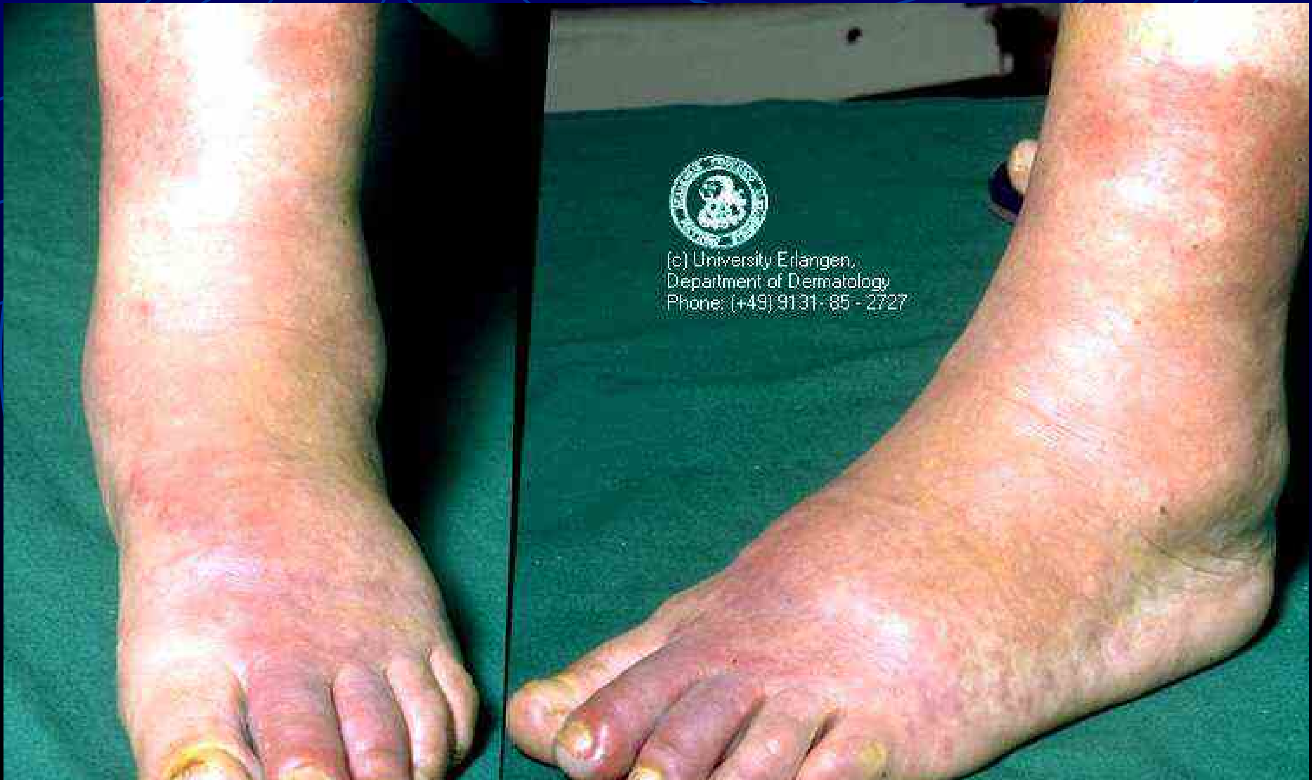
  Supportive care including rest, leg elevation, sterile dressings and analgesia
- *Complications : gangrene , Local necrosis, abscess and septicaemia*



Erysipelas

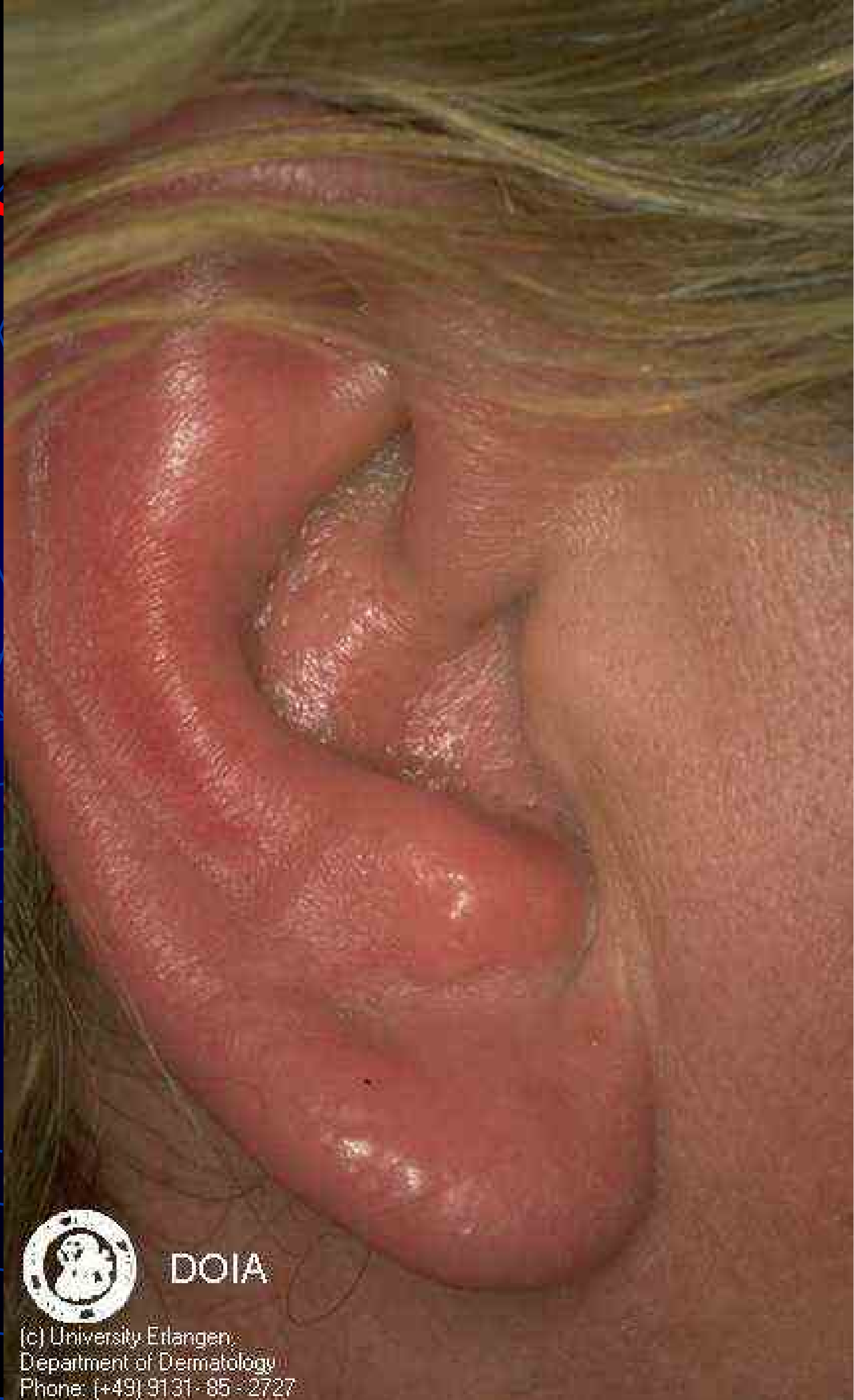


Cellulitis with elephantiasis of the penis


(c) University Erlangen,
Department of Dermatology
Phone: (+49) 9131-85-2727

What is your diagno

- A. Erysipelas
- B. Cellullitis



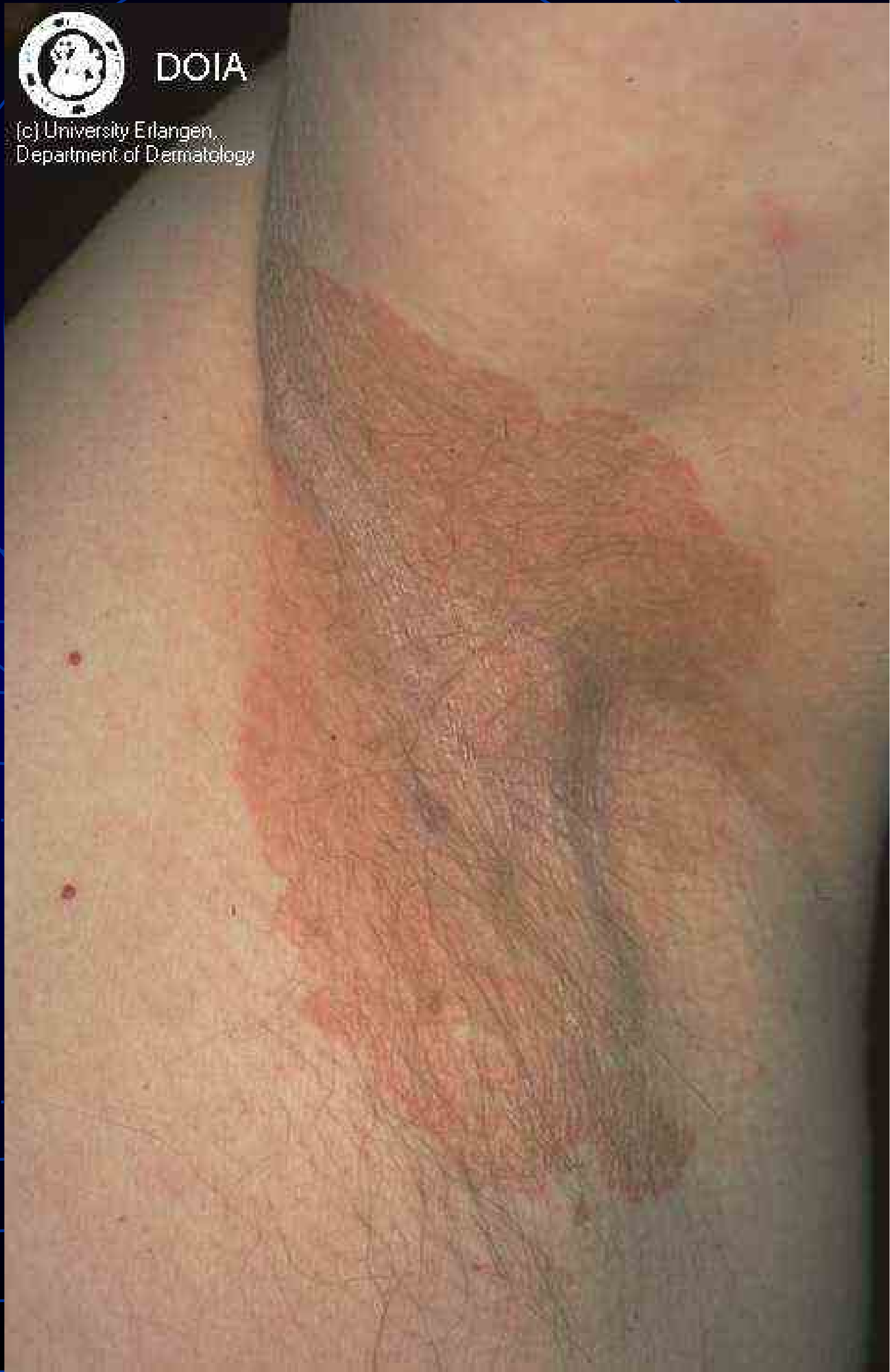
DOIA

(c) University Erlangen,
Department of Dermatology
Phone: (+49) 9131 - 85 - 2727

Erythrasma

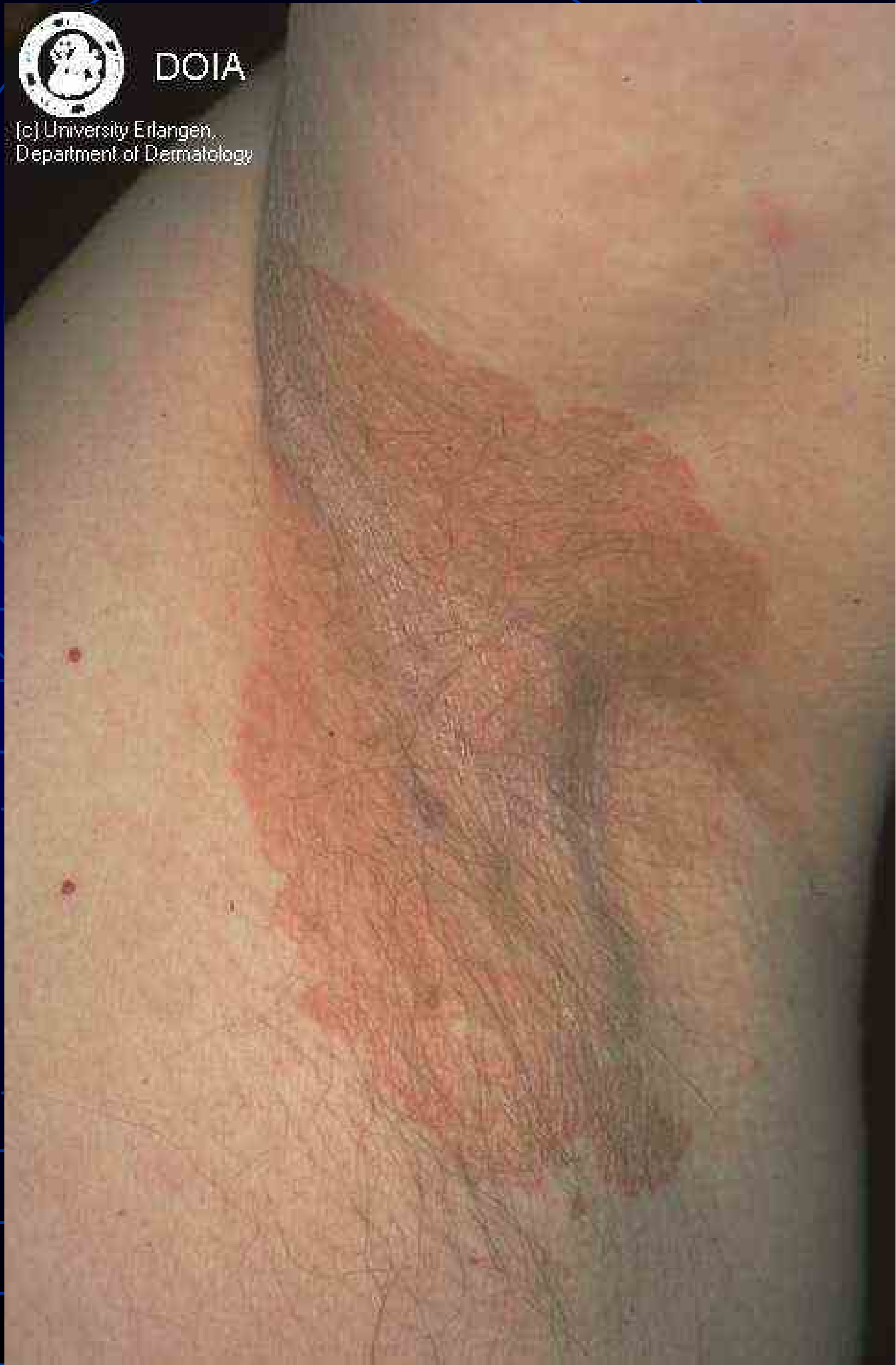
- Erythrasma is a mild, chronic, localized superficial infection of the skin caused by the diphtheroid *Corynebacterium minutissimum.*

- These are aerobic, gram positive, pleomorphic, rods.

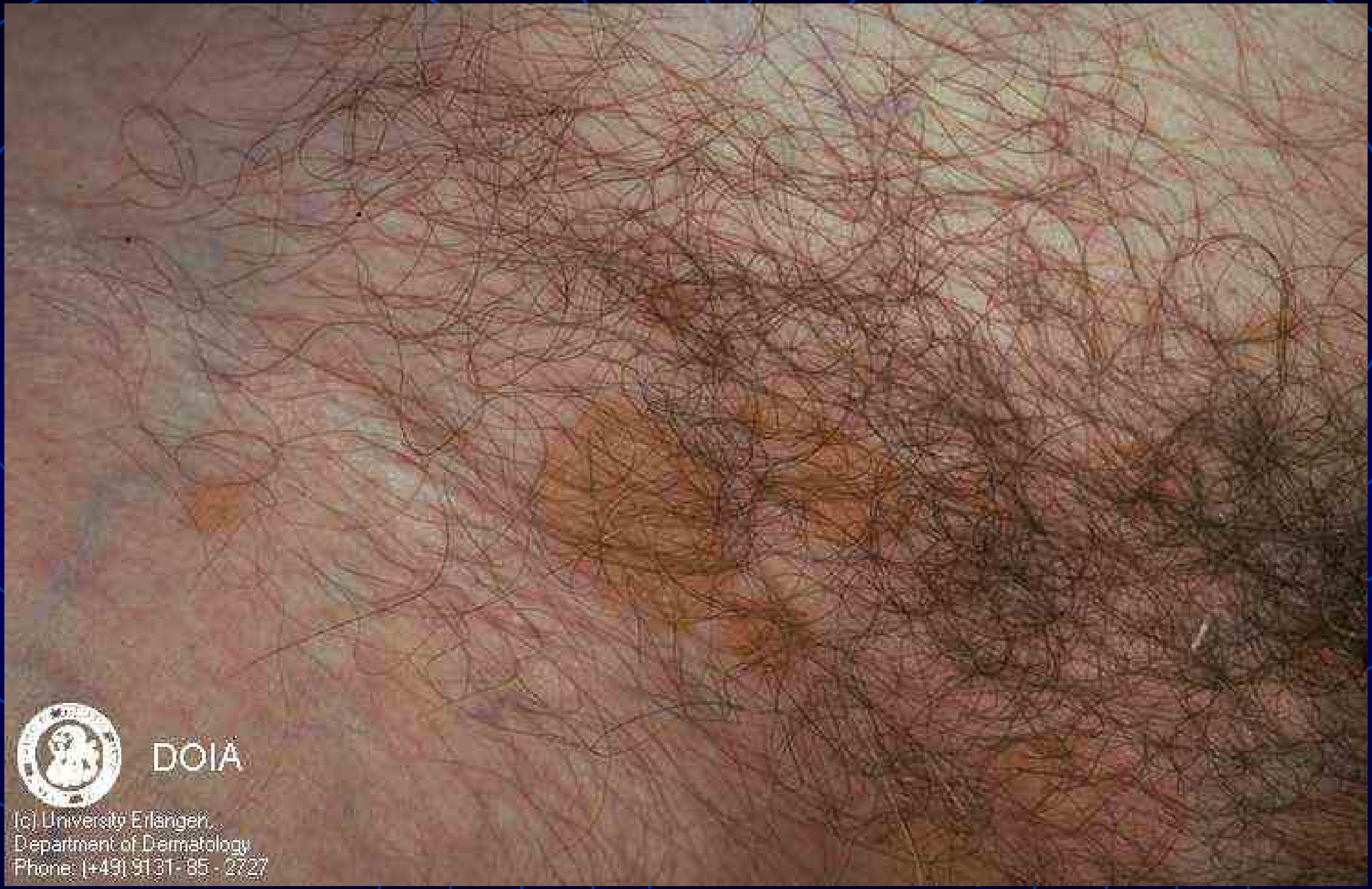

DOIA
(c) University Erlangen, Department of Dermatology

Erythrasma

- It is characterized by sharply marginated, dry, brown, slightly scaling patches occurring in the intertriginous areas, especially the groins, axillae and the intergluteal and submammary flexures. Erythrasma is more common in adults. A warm, humid climate and diabetes are predisposing factors.

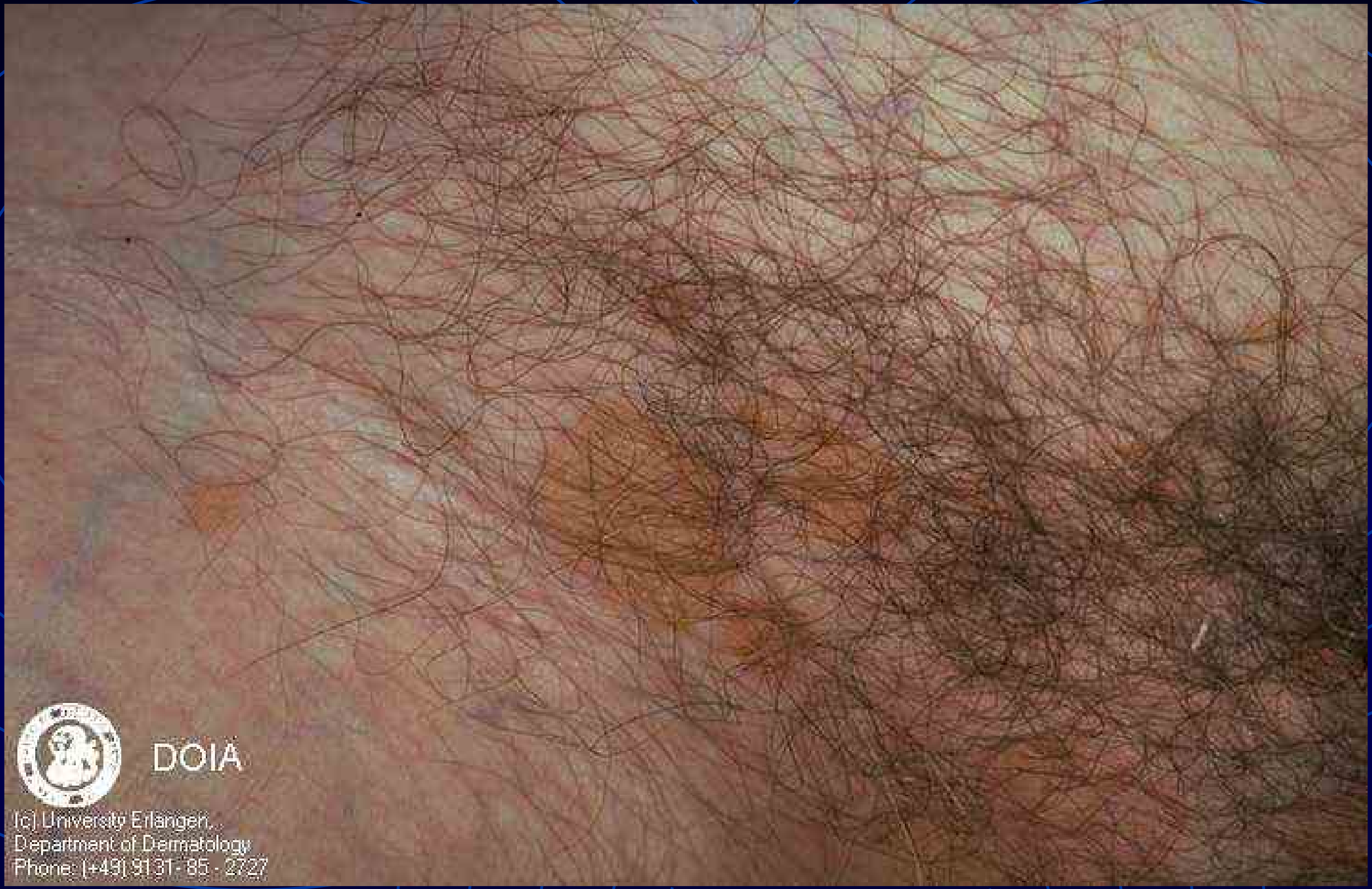

DOIA
(c) University Erlangen, Department of Dermatology

Erythrasma


DOIA
(c) University Erlangen,
Department of Dermatology
Phone: (+49) 9131-85-2727

- Wood's light examination is diagnostic for erythrasma, the affected area shows a coral red fluorescence.

Erythrasma

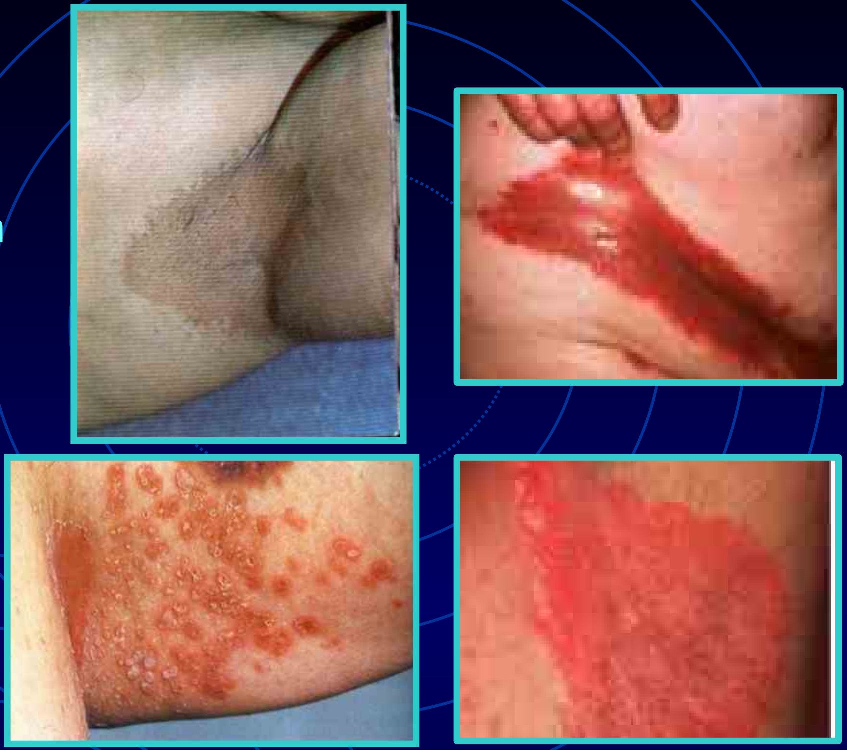

DOIA
(c) University Erlangen,
Department of Dermatology
Phone: (+49) 9131 - 85 - 2727

- ***Treatment***

Topical azole antifungals.

Oral erythromycin in extensive cases.

INTERTRIGO



Definitions: intertrigo is inflammation of the flexural skin

Causes

A- Infective
- (1)streptococcal
- (2)fungal.
  - a-cndidial
  - b-tenia cruris
- (3) Erythrasma

B- Non- infective
- (1)simple (friction)
- (2)seb. dermatitis.
- (3)Psoriasis

Angular cheilitis

-It is the inflammation of the mouth angles

-Causes:
- (1) streptococcal
- (2) Candida
- (3) Vit. B deficiency

Note

Pseudo- folliculitis

It is the inflamation of skin of beard and results from re- entering of hairs to skin with shaving . →Irritation →papules.

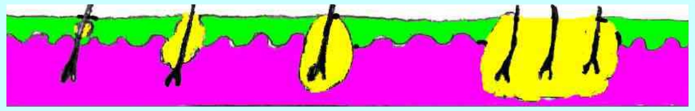
Cutaneous involvement in systemic sterpt. Infecions

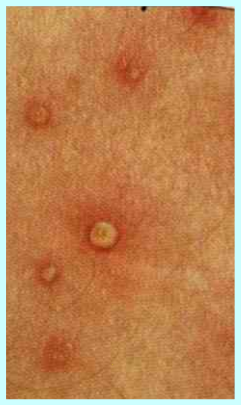
1-Rash of scarlet fever.

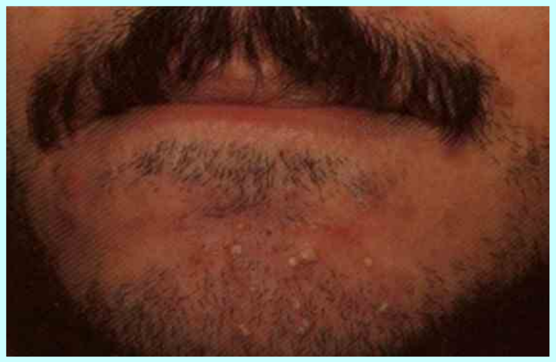
2-Petechae, sub- ungual (splinter) haemoshages in subacute bacterial endocardidtis .

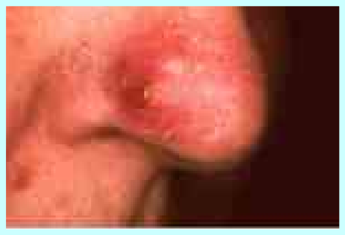
3-Erythema nodosum .

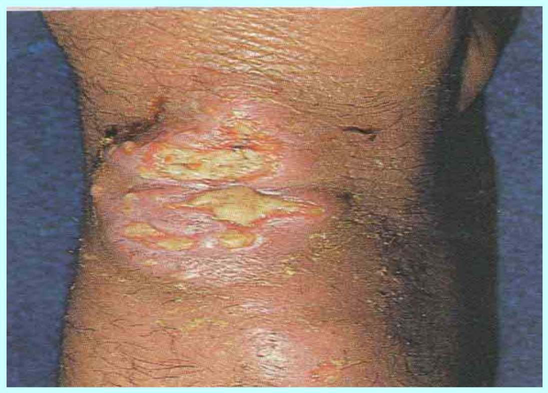
4-Erythema multiform .

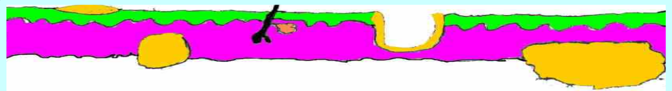


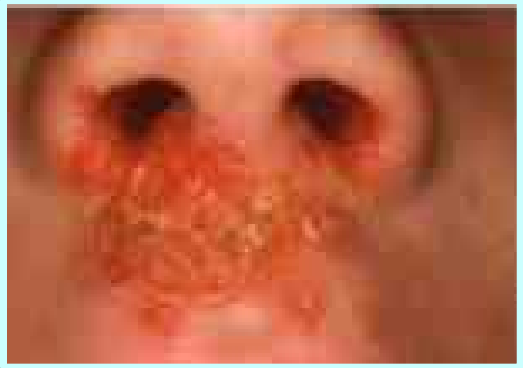


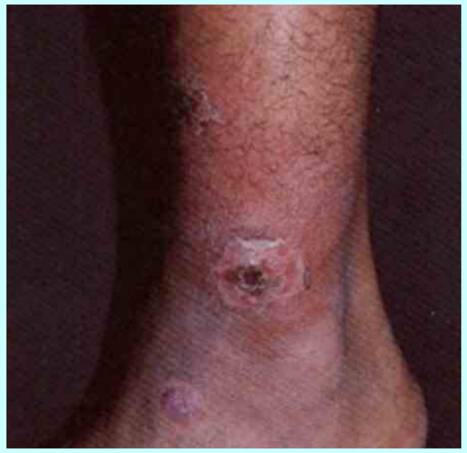


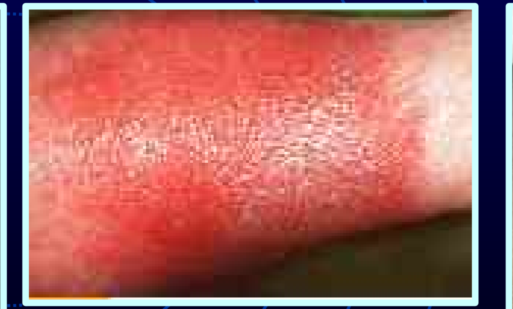


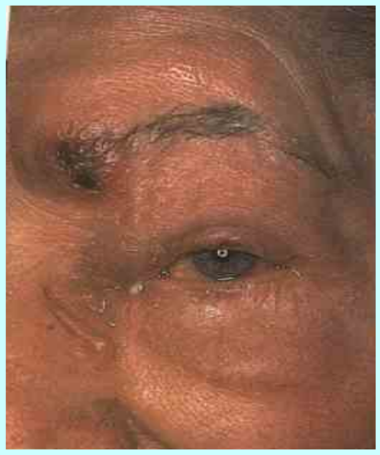












viral INFECTIONS

Dr Abdelwassa Almekhlafi, MD

Characters of the viruses:

-Obligate intracellular, multiply only inside the cells (outside the cells presents as particles (virions).

-Composed of single nucleic acid : DNA or RNA.

-Doesn’t grow on ordinary media .

-Not sensitive to antibacterial drugs.

-Generally are not thought to exist on the skin surface as commensals.
-Differ from other replicating organisms in that they donot have ribosomes or enzymes for high energy phosphate generation or for

protein, carbohydrate, or lipid metabolism

The Consequences of viral infections

viral	cellular
-attachment.	1 -cytolysis (cell death) e.g.
-penitration	-chicken pox
-removal of the caspid.	-Herpes S, H. Z.
-replication.	-small pox
-release.	2-neoplastic transformation. e. g. warts.
	3- latency

Host-response to viral Infection:

(1)-Humeral (important against reInfection):-

a-neutralization
b-enhancement of viral uptake by phagocy tocytes
c-complement

(2)-Specific cell-mediated:-

Important for influencing the course of many viral infections

DNA viruses

- -Papova virus: papilloma V
- -Human herpes viruses (HHVs)
  - HHV1
  - HHV2
  - HHV3 (varicella zoster virus)
- -Pox viruses
  - vaccinia
  - orf
  - molluscum contagiosum

RNA viruses

- Togavirus
    Robella
-Retroviruses
    HIV1
      HIV2

VIRAL INFECTIONS

The main viral diseases of the skin are:

- Herpes simplex.
- Herpes zoster.
- Warts.
- Molluscum contagiosum.

Herpesvirus group

The herpesvirus group are DNA viruses that replicate intranuclear.

They are characterized by the absence of virus elimination following clinical recovery.

Virus persists throughout the person’s life as a latent infection in one of the sensory ganglia.

Under certain conditions, especially immune suppression, the virus may become replicated and produce infection.

Herpes simplex

- It is one of the most common infections of the humans throughout the world.

Etiology Herpes simplex is caused by . *Human herpes virus type 1 ,2.*

There are two types of HSV:

Type I, the cause of facial infections.

Type II, the cause of genital infections.

The spread of infection is by direct contact, or droplets, from infected secretions. Incubation period: 2-12 days.

Clinical picture

- Primary herpes simplex

Primary infection occurs in individuals who are infected for the first time. It is usually subclinical, but when clinical lesions develop, the severity is generally greater than in recurrences.

Primary infection occurs mainly in infants and young children.

Constitutional symptoms as fever and malaise are common. Vesicles show no tendency to grouping and

Clinical types

Herpetic gingivostomatitis. It involves the gums, tongue, pharynx, palate, and buccal mucous membrane.

Herpes genitalis. It occurs after the onset of sexual activity and affects the glans, prepuce and shaft of the penis in males, and the vulva, vagina and cervix in females.

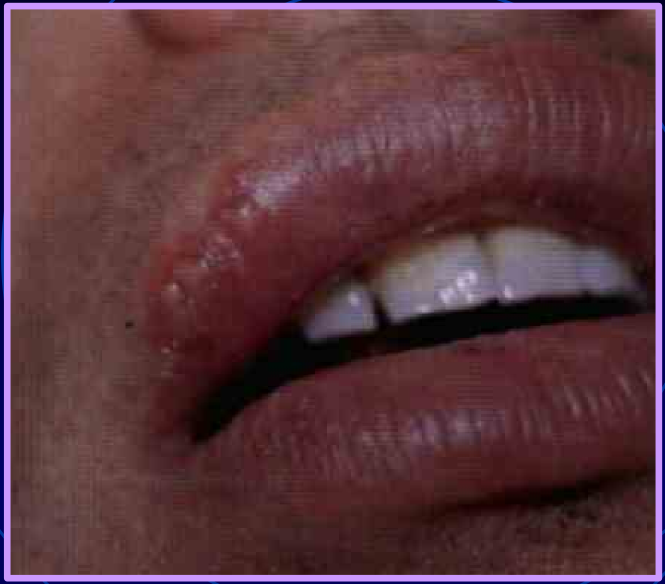
Keratoconjunctivitis. It manifests as keratitis, conjunctivitis and edema of eyelids.

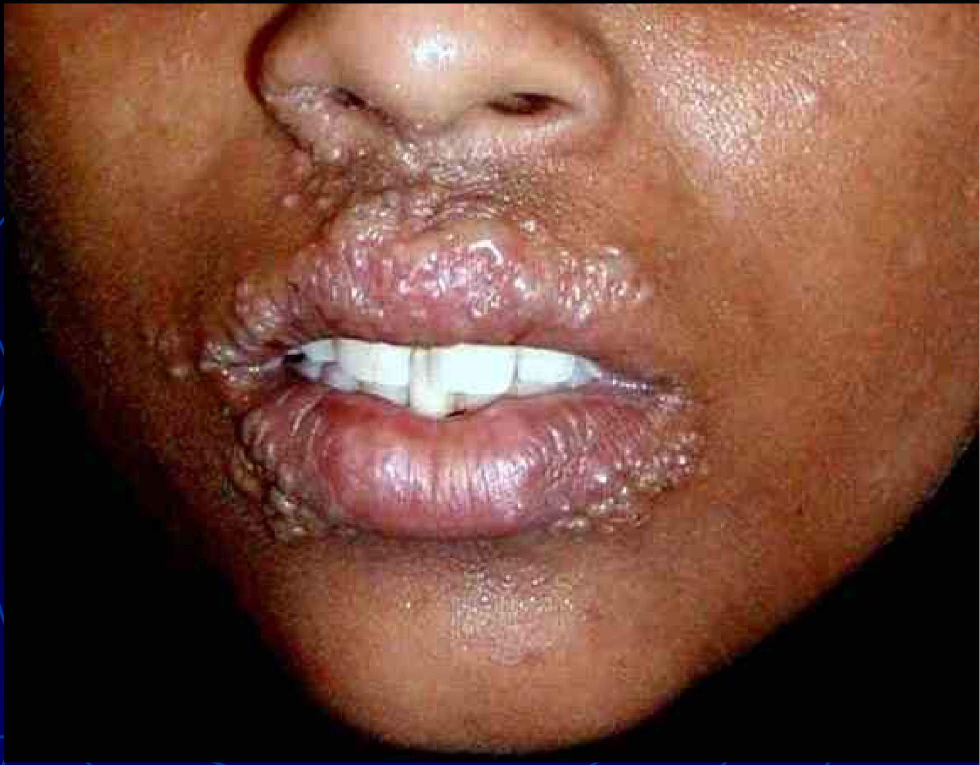
Inoculation herpes simplex. It occurs secondary to direct inoculation of the virus into the skin.

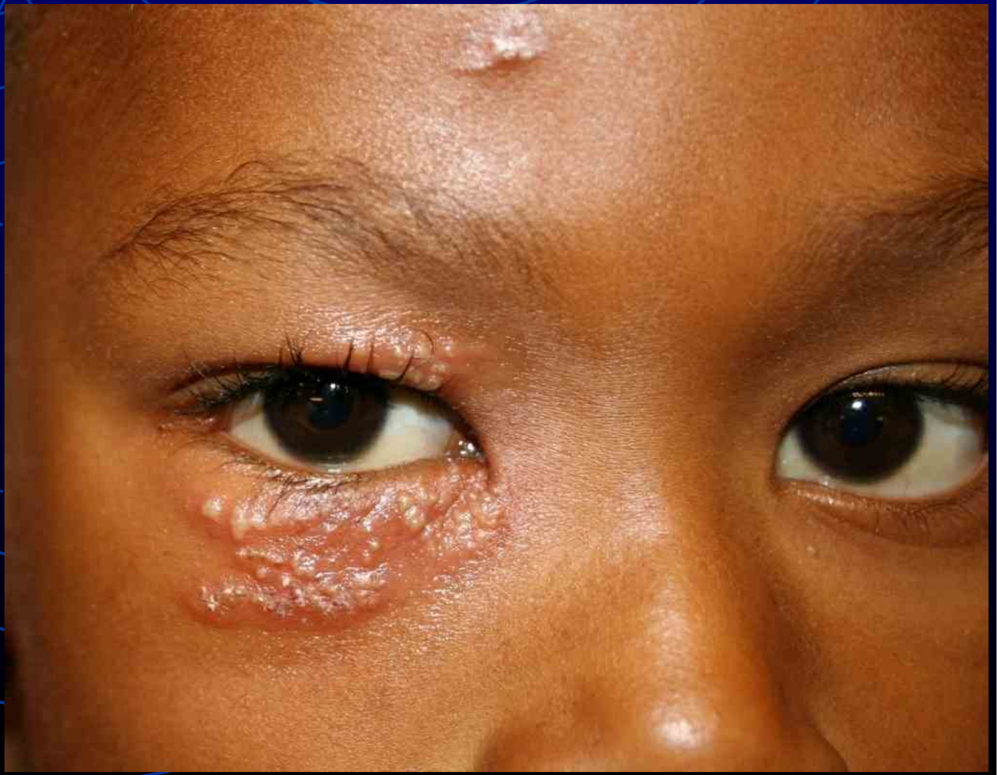
Eczema herpeticum (Kaposi's varicelliform eruption). The infection occurs in many skin diseases particularly atopic dermatitis.

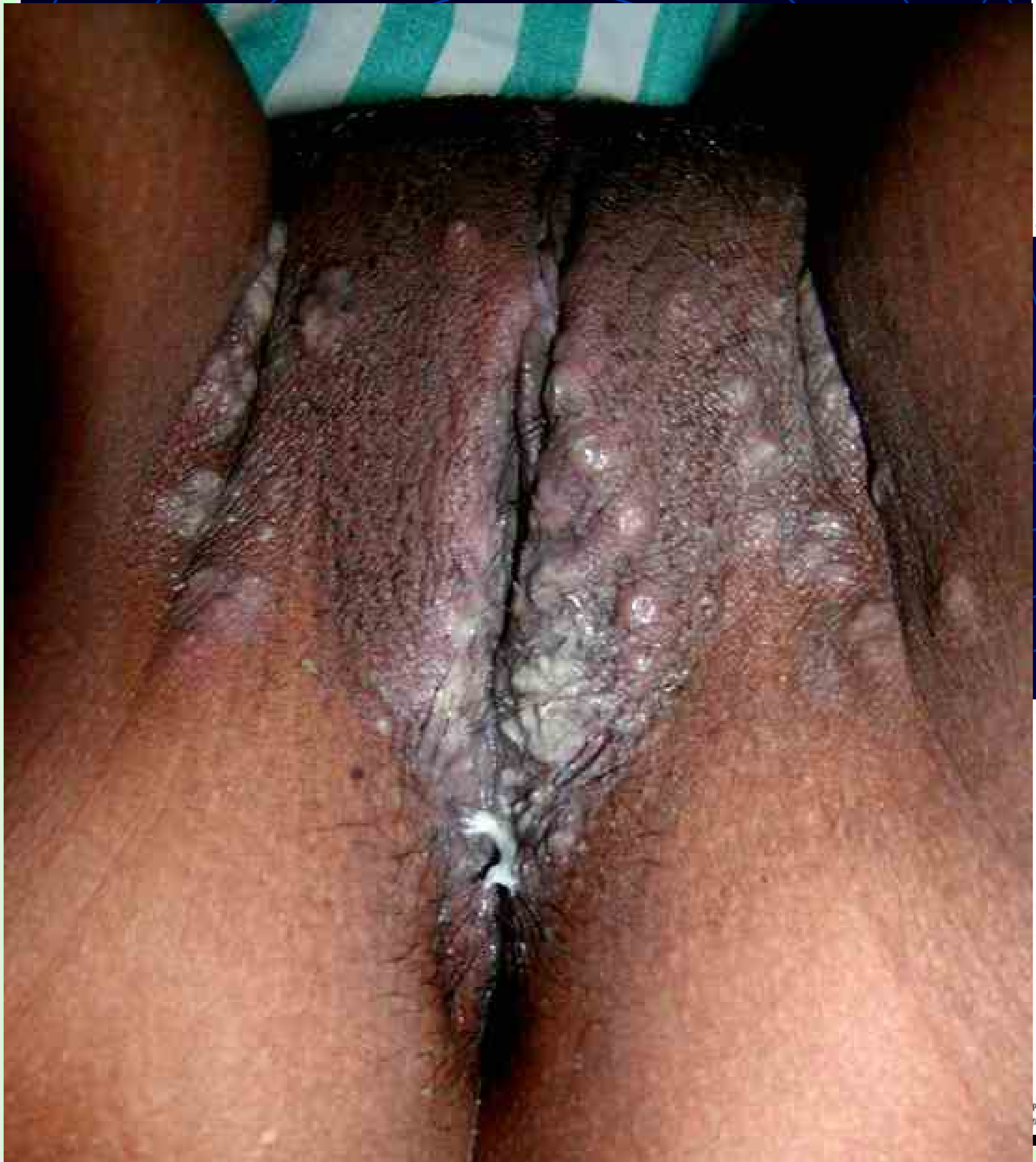
Neonatal herpes simplex. It is acquired as the child passes through an infected birth canal of the mother. In these cases cesarean section or treatment with acyclovir are indicated

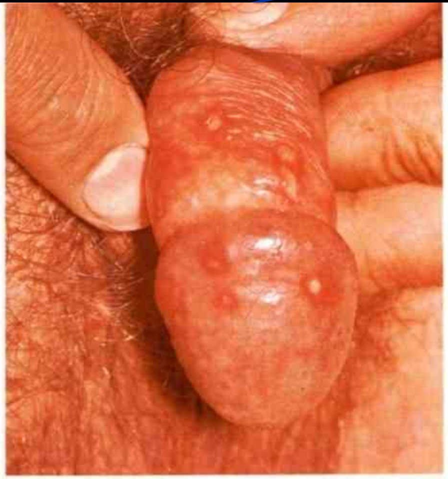
Herpes simplex



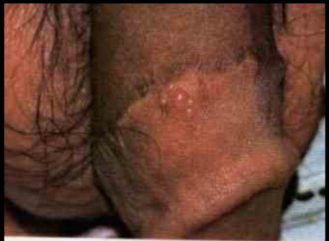


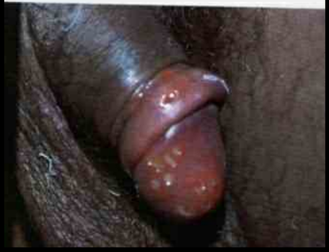


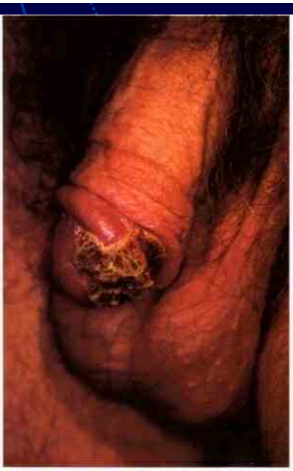




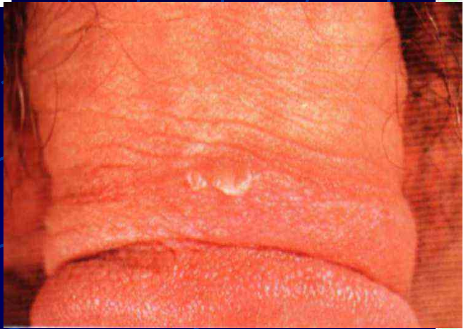
Recurrent genital herpes simplex infection *Characteristic grouped, painful erosions on the glans penis and foreskin.*

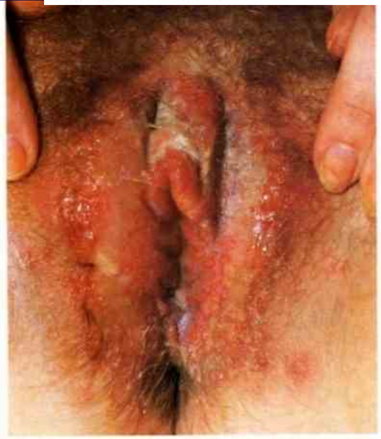




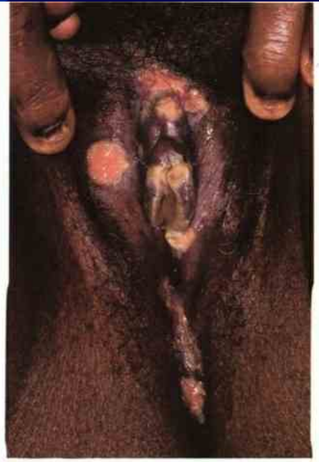


Chronic herpetic ulcer *A large crusted ulcer is seen on the distal shaft and glans penis. The lesion had been present for 1 year. The patient was being treated with intravenous phosphonoformate and healed completely in 3 weeks.*





Primary herpetic vulvitis *Confluent, tender erosions with exudation associated with very painful inguinal lymphadenopathy.*



Chronic herpetic ulcers *Large ulcers are seen on the vulva and perineum. The lesions cleared completely on oral acyclovir.*

Recurrent herpes simplex

It occurs in persons previously infected with the virus. If the virus is reactivated, it may travel peripherally along the nerve fibre, and, if it replicates in the skin and mucous membranes, may cause recurrent disease.

Triggering factors

- * Minor trauma * Immunodeficiency * Ultraviolet radiation * Neural or dental surgery * Emotional stress * Menstruation

Recurrent infections differ from primary infections in the smaller size of the vesicles and their close grouping, and in the usual absence of constitutional symptoms.

The onset is sudden, often preceded by itching or burning. A swollen, red patch, 1-2 cm in diameter, appears and soon grouped small vesicles on an erythematous base develop. In one or two days the vesicles dry with the formation of

Clinical types

- **Herpes facialis (labialis).** Favorite sites are the lips, around the nose and cheeks. Recurrences tend to be in the same region.

- **Herpetic keratoconjunctivitis.** It occurs as a punctate or marginal keratitis, or as a dendritic corneal ulcer.

- **Herpes genitalis.** It usually presents with clusters of small vesicles, which produce non-indurated ulcers on the glans, or shaft of the penis. Similar lesions may occur on the labia, vagina or cervix.

Treatment

Topical treatment

- Drying lotions as 5% aluminium acetate for the vesicular stage.
- Antibiotics for secondary infection.
- Acyclovir cream.

Systemic treatment

- It is used only in severe cases.
- Acyclovir is the treatment of choice.
- New antivirals e.g. valacyclovir and famciclovir are also effective.

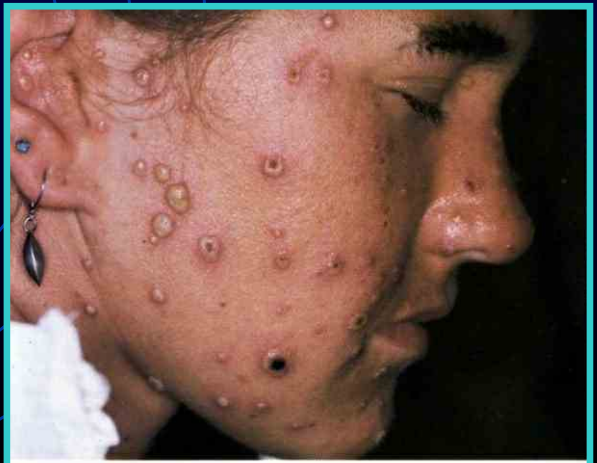
Varicella (Chickenpox)

- Varicella is the primary infection with *Varicella-zoster virus*. **I.P.** is 10-21 days. Transmission is by direct contact with the lesions or by the respiratory route. One attack usually confers permanent immunity. Individuals are infectious for 4 days before and 5 days after the appearance of the exanthem.

- Low-grade fever, malaise, and headache are usually present but mild. The eruption starts with faint macules that develop rapidly into vesicles. Successive fresh crops of vesicles appear for few days, mainly on the trunk, face and oral mucosa (centripetal). The vesicles quickly become pustular, umbilicated, and then crusted. Lesions in different

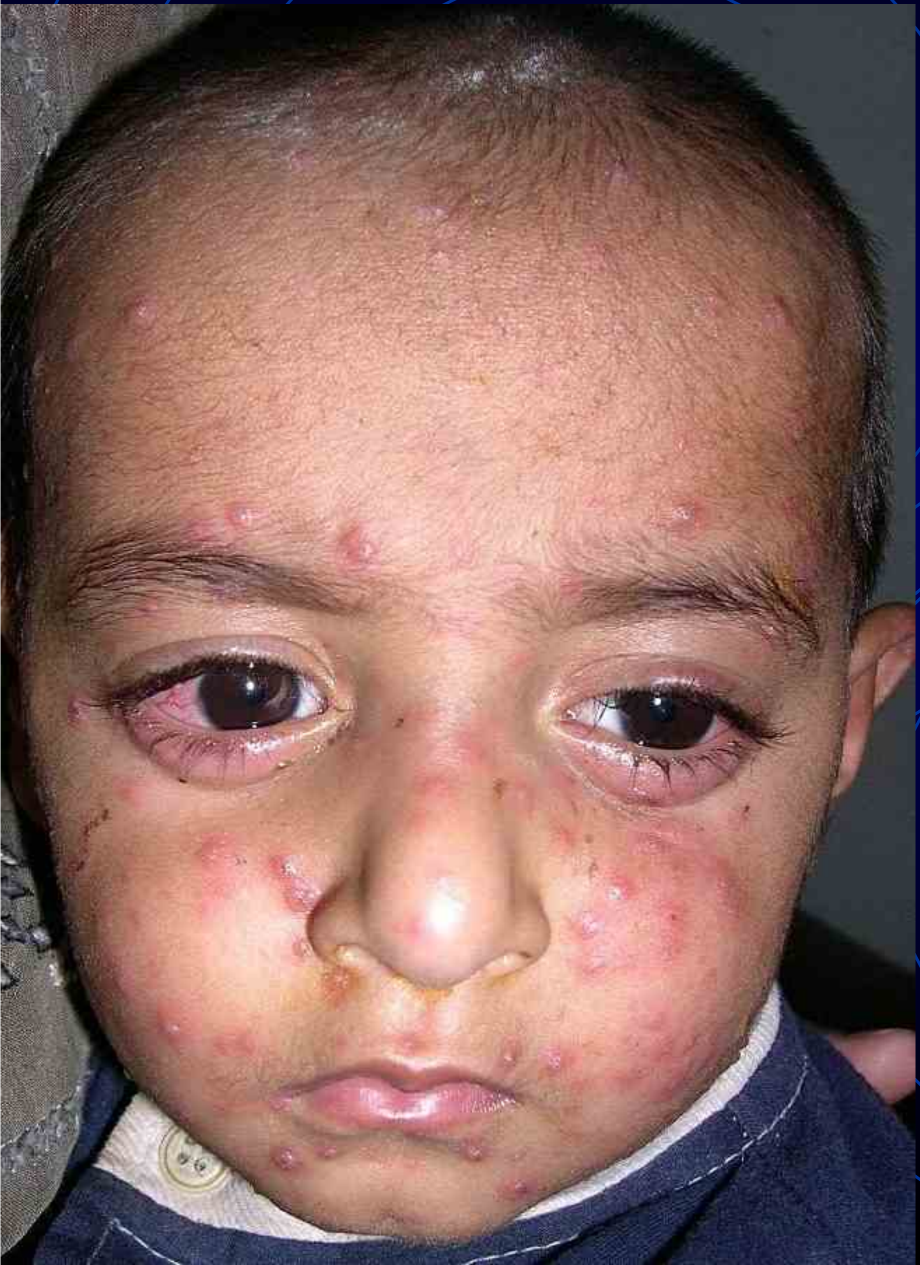
Complications:

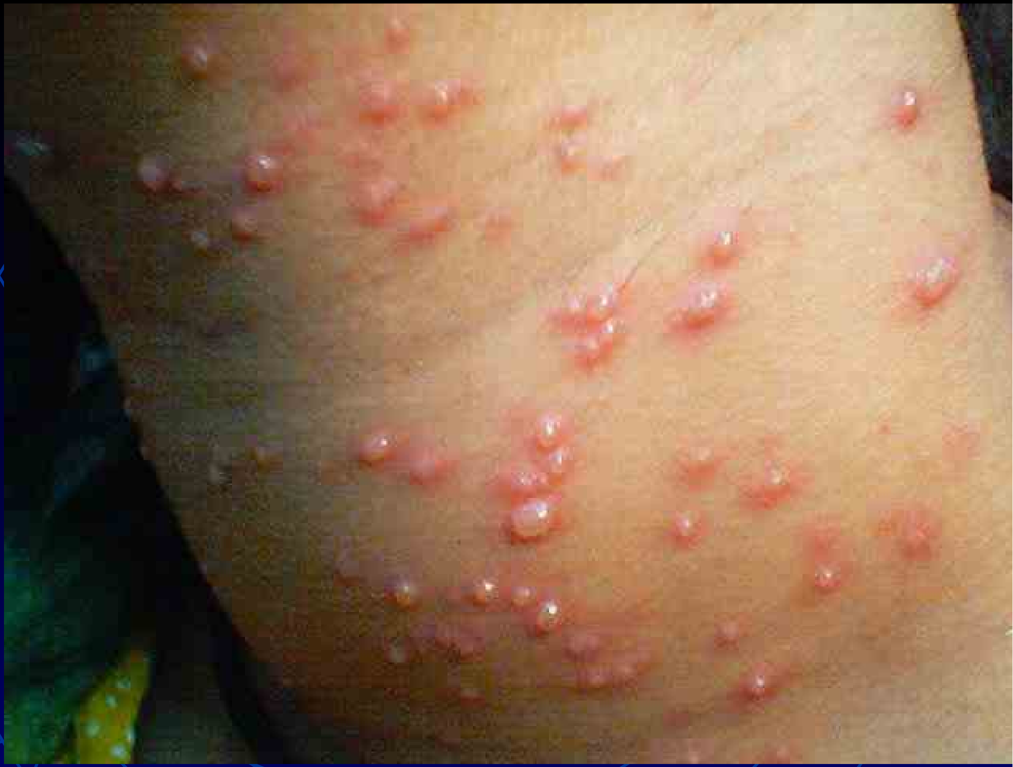
- Bacterial skin infections
- neonatal < 5 d => neonate CH. P
- Fetal varicella when acquired early in pregnancy (first 20 weeks) Low birth weight, mental retardation, seizures, skeletal problems, dermatomal hypoplasia
-varicella pneumonia
- keratitis
- Encephalitis
- Reye syndrome: fatal incephlopathy with varicella
- intrauterine 1st trimester => zosteriform cutaneous scar and limb hypoplasia.

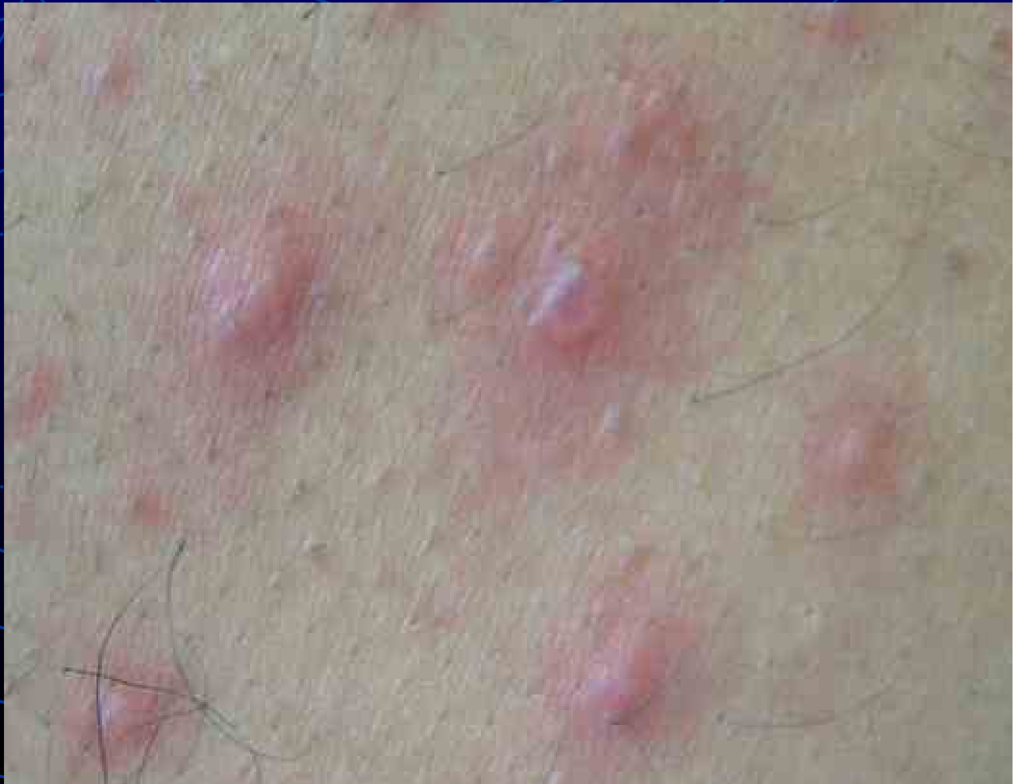


7 Varicella *Discrete vesicles, pustules, and crusted lesions are seen simultaneously.*

Chickenpox

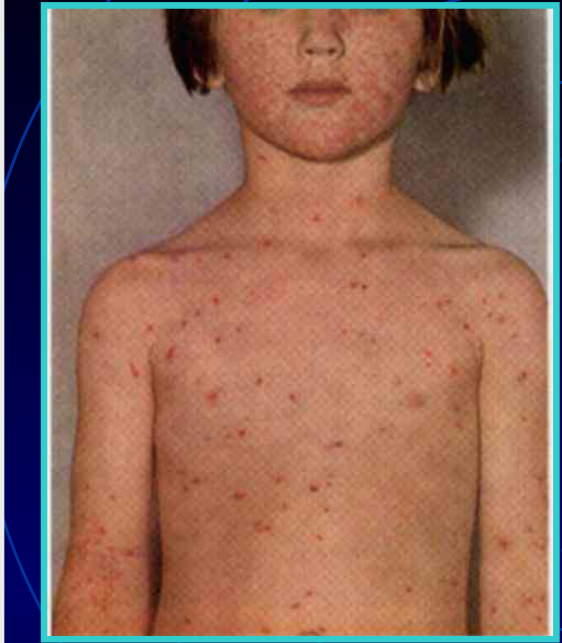


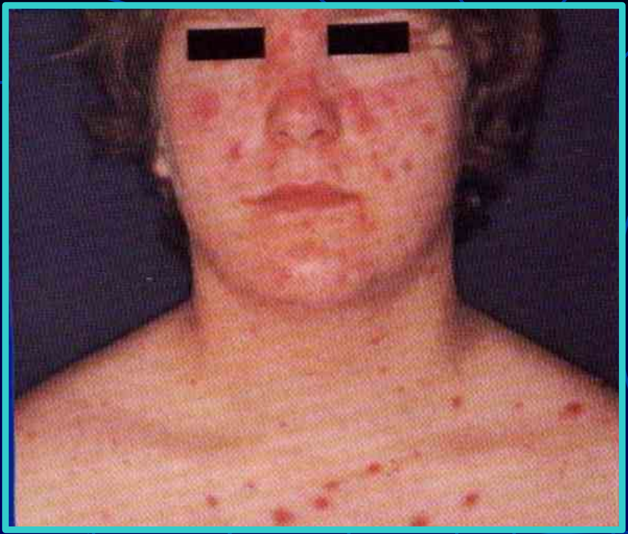


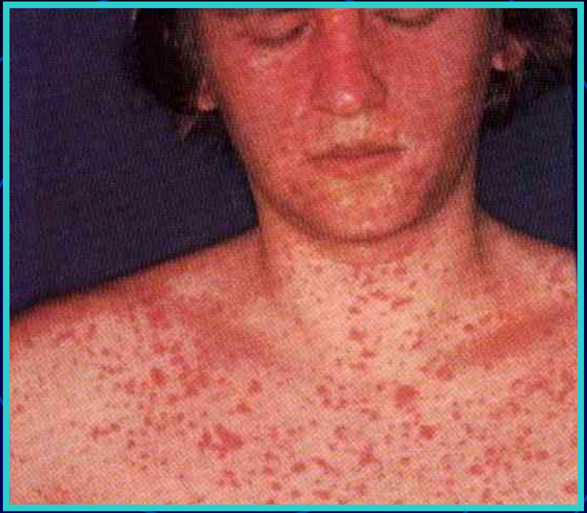


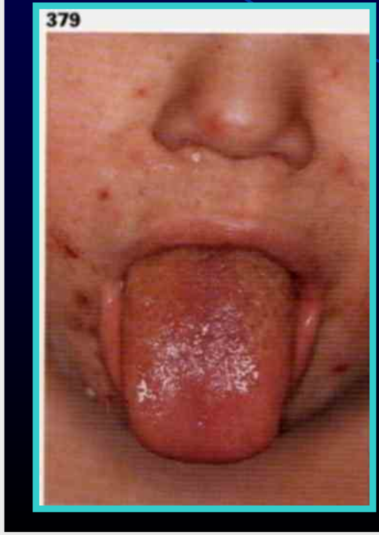
Treatment

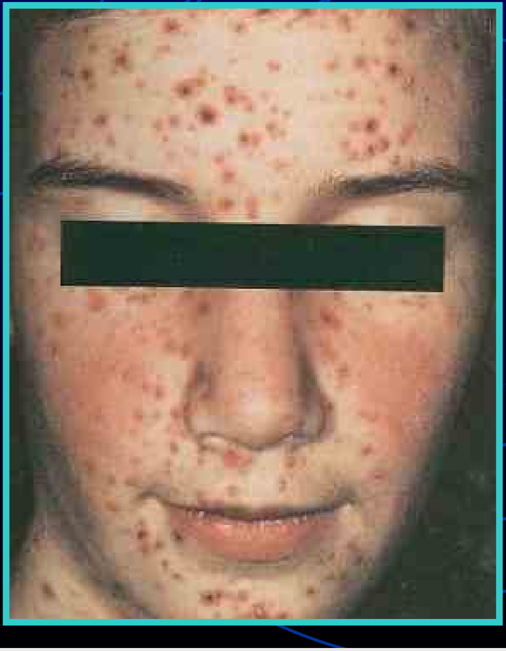
- Treatment is symptomatic. Antipruritic lotions and anti-histamines are given for itching and antibiotics for secondary infection.
- Acyclovir 800 mg four times daily for 7 days.



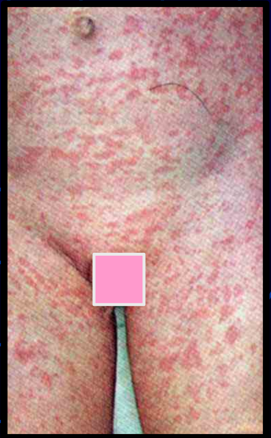


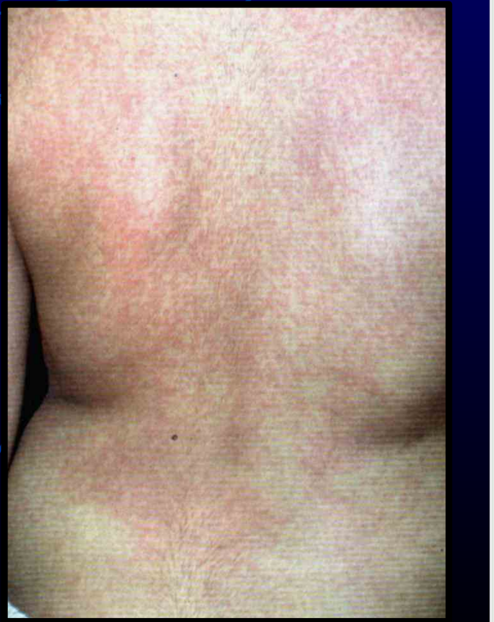






Measles





Herpes zoster

H.Z. is an acute skin infection characterized by an eruption of grouped vesicles following the distribution of a peripheral nerve (usually sensory). The disease is also more frequent in immunocompromised persons.

Etiology

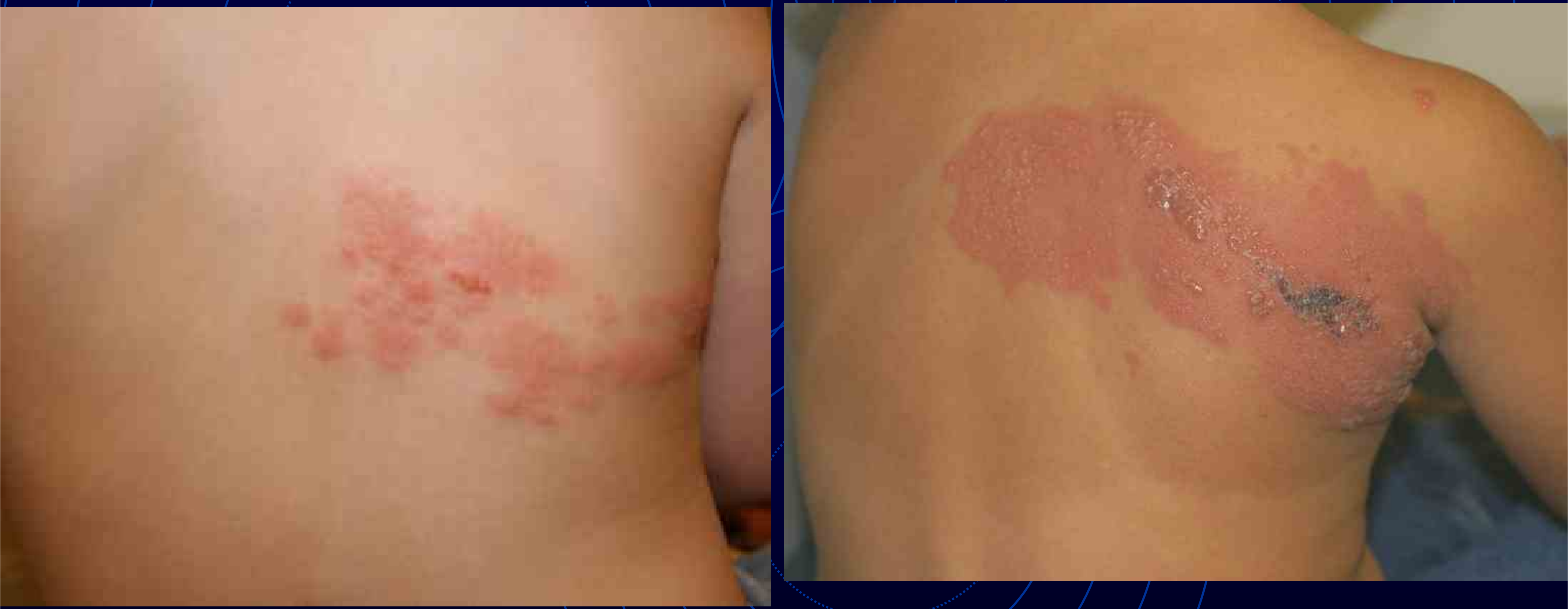
- *Varicella-zoster virus* is the cause of the disease as a result of reactivation of the latent virus. Other than immunosuppression and age, the factors involved in reactivation

Clinical picture

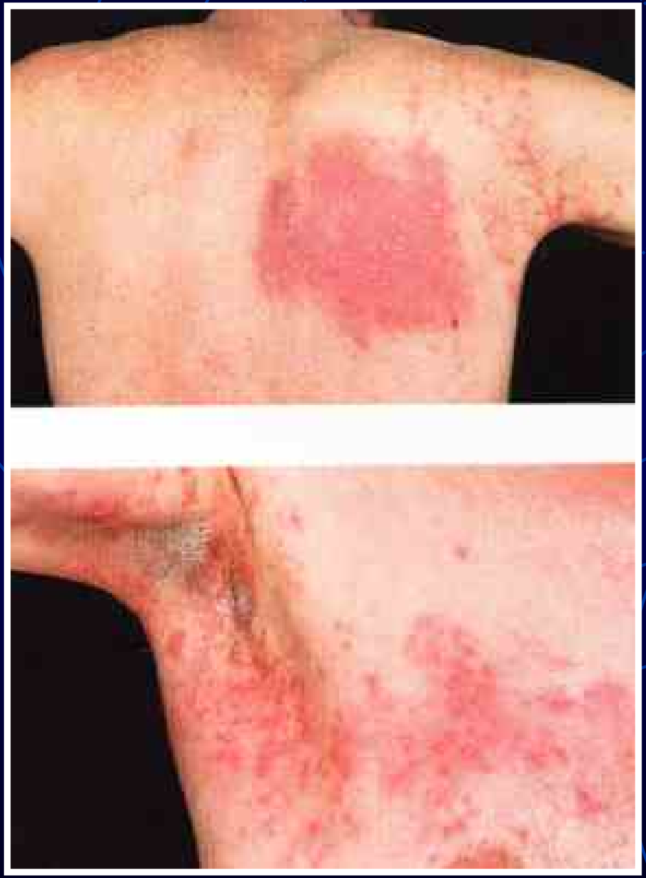
- Pain is usually the first manifestation of the disease, which may be severe. Pain may appear before, simultaneously or following the skin eruption, or may be absent (children).

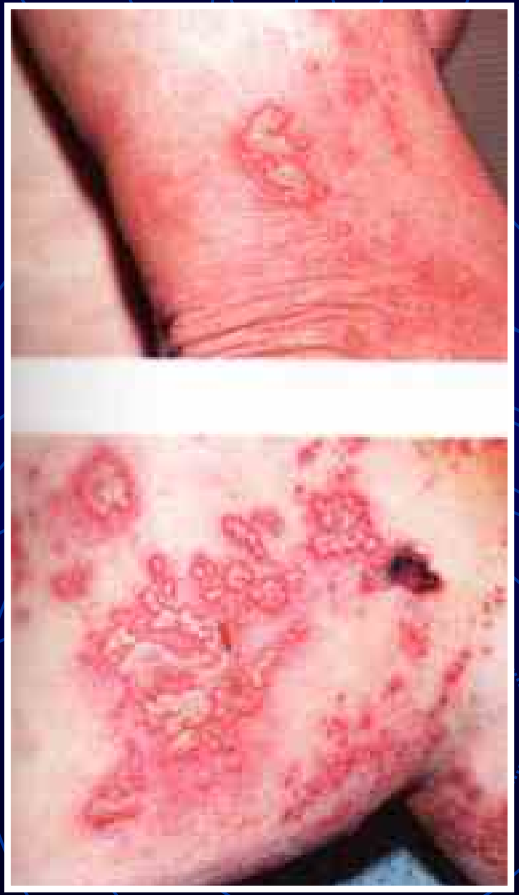
- After 2-3 days, the eruption appears as groups of vesicles on an erythematous base within the distribution of a sensory nerve. The vesicles become pustular, crust and heal in 2-4 weeks with scars. The regional lymph nodes are enlarged and tender.
- The eruption is almost invariably unilateral and usually affects one dermatome.
- The thoracic (commonest), cervical, trigeminal and lumbosacral dermatomes are the most commonly affected.

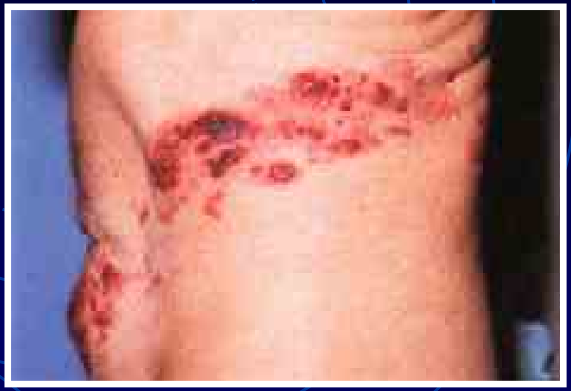
Herpes zoster

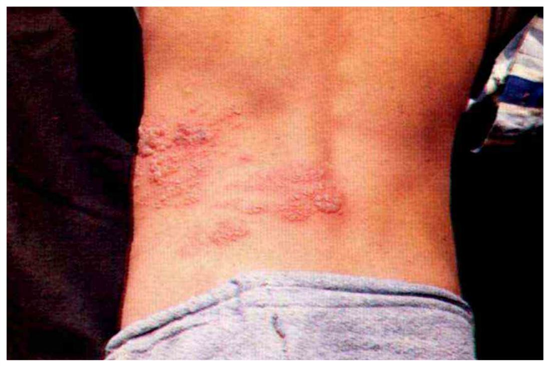


Herpes Zoster (H.Z)









Ophthalmic zoster (Herpes zoster ophthalmicus)

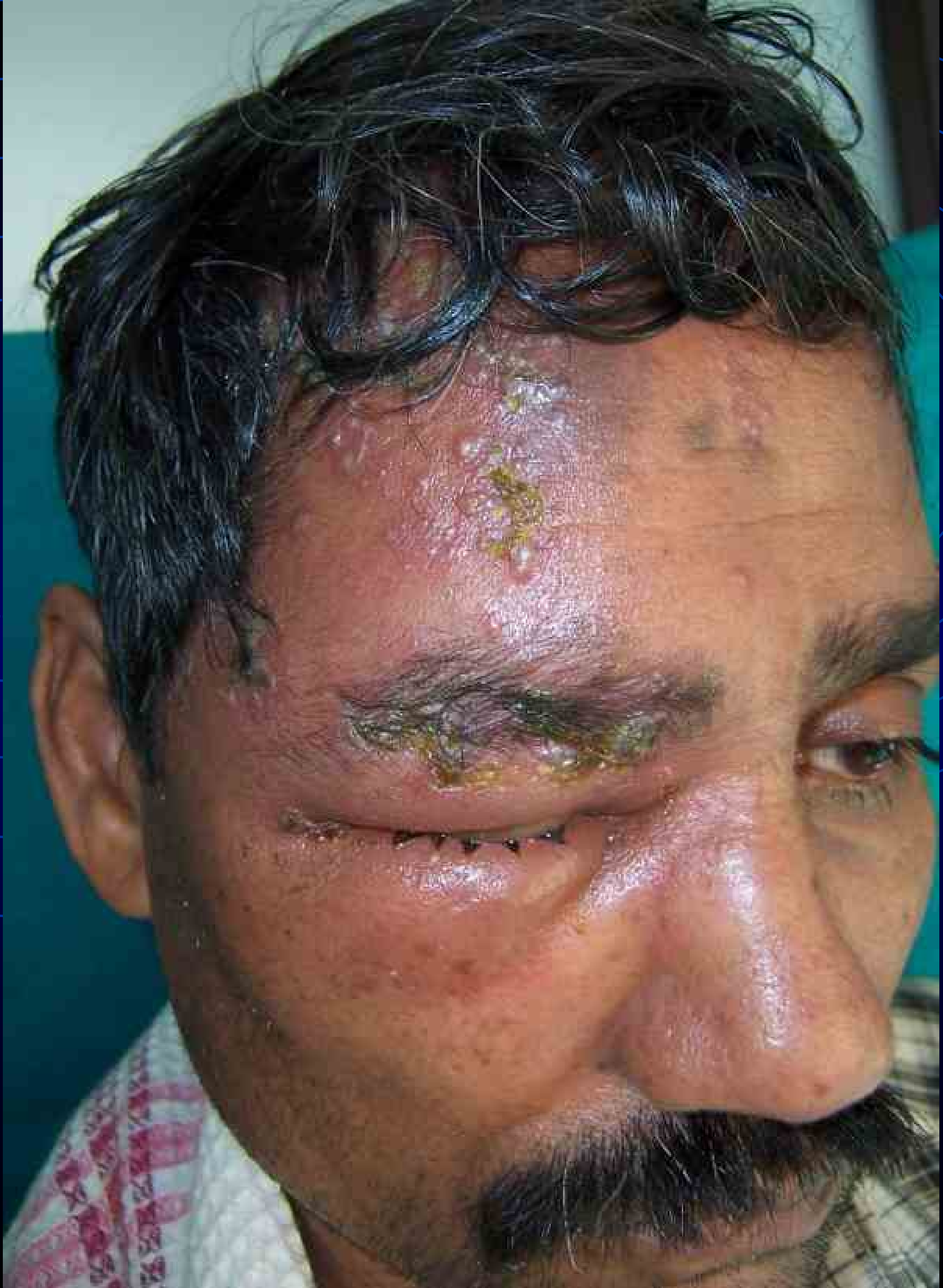
- Herpes zoster may affect any of the divisions of the trigeminal nerve, but the ophthalmic division is the most frequently involved. Ocular involvement is most commonly in the form of uveitis and keratitis.

Ramsay-Hunt syndrome

- It is due to involvement of the geniculate ganglion and constitutes vesicles on the external ear, facial palsy and auditory symptoms as tinnitus, deafness and vertigo.

Complications

- Post-herpetic neuralgia. It is the commonest and the most intractable complication of herpes zoster. It can be defined as the persistence of pain more than one month after the onset of the disease.
- Ocular complications. Secondary bacterial infection and gangrene.
- Encephalitis Facial palsy



Treatment

Topical treatment

- *As for herpes simplex.*

Systemic treatment

- *Analgesics for pain.*
- *Antibiotics for secondary bacterial infection.*
- *Acyclovir and the newer antivirals e.g. valacyclovir and famciclovir are used in*

Warts (Verrucae)

Mode of transmission and Clinical types. Warts are common contagious cutaneous growths caused by *Human papilloma virus* (HPV). Warts spread by direct or indirect contact. Trauma and maceration greatly predispose to inoculation of the virus.

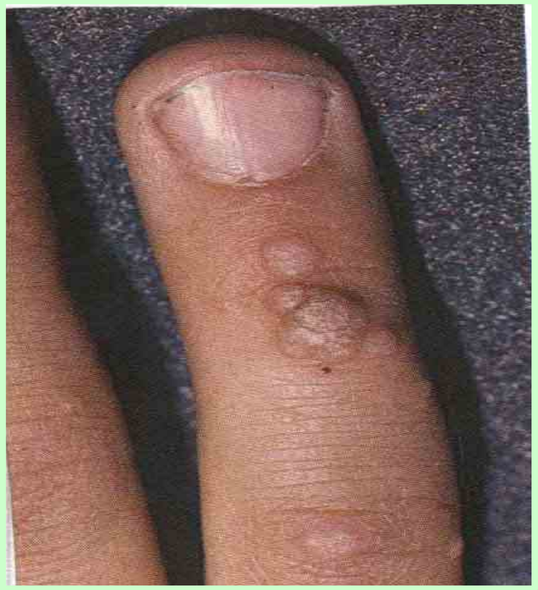
Common warts. They appear as papules with rough, horny surface. The lesions are usually symptomless.

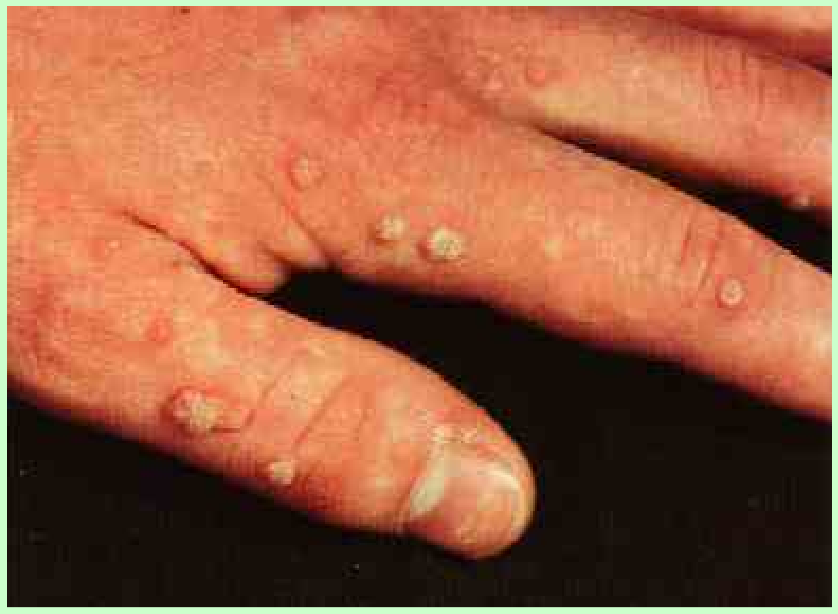
Plane warts (flat warts). They are smooth, flat or slightly elevated lesions and are usually skin-colored or greyish yellow. The lesions vary from 1-5 mm. The face and back of the hands are the sites of predilection. New warts may form at the site of trauma (Koebner's Phenomenon).

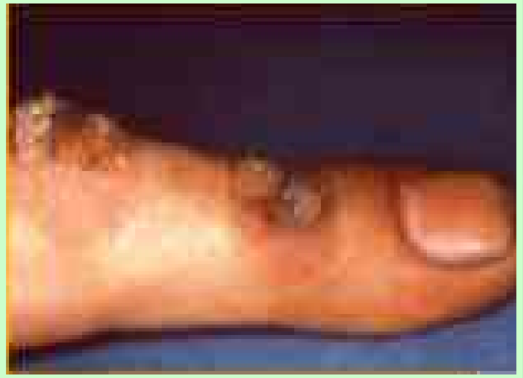
Filiform warts. **Digitiform warts.**

Plantar warts. These affect the sole of the foot especially the pressure points and presented as sharply defined, rounded, slightly elevated lesions with a rough keratotic surface. Pain is a common symptom.

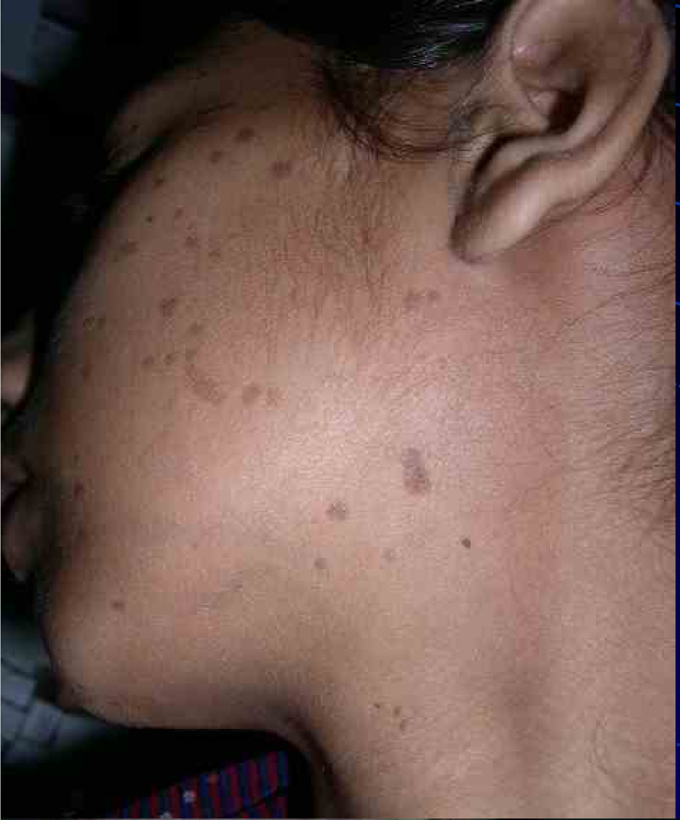
Anogenital warts (condylomata acuminata). Genital warts are the most common sexually transmitted disease. The wart is soft, pink, elongated and sometimes pedunculated. The lesions are usually multiple especially on moist surfaces. The sites of predilection are frenulum, corona and glans in men, the posterior fourchette in women and perianal area and groins in both sexes. Anogenital warts are usually asymptomatic, but sometimes can cause discomfort, discharge or bleeding. They are

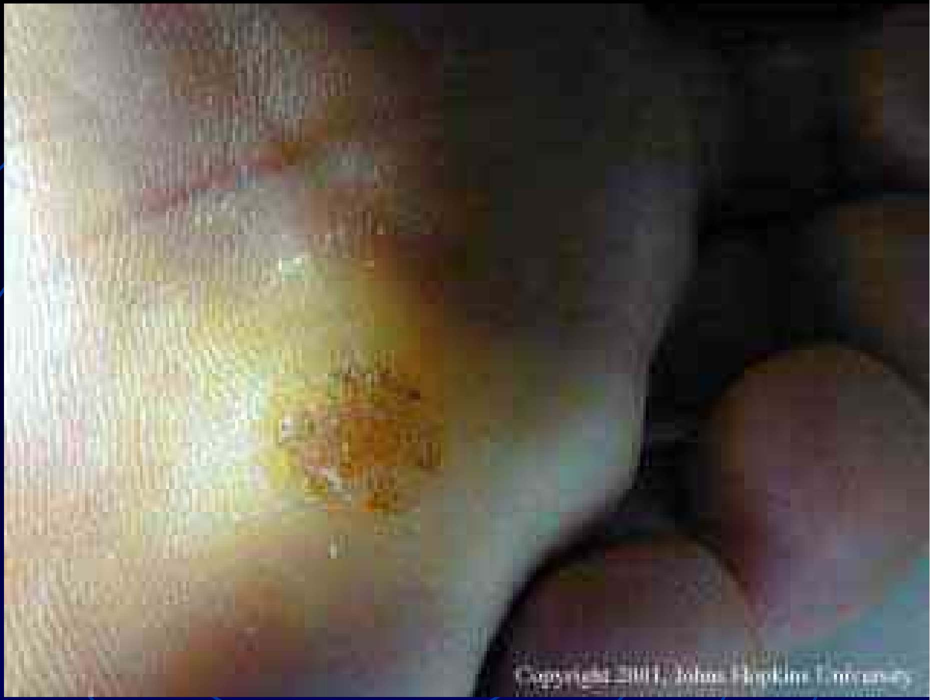


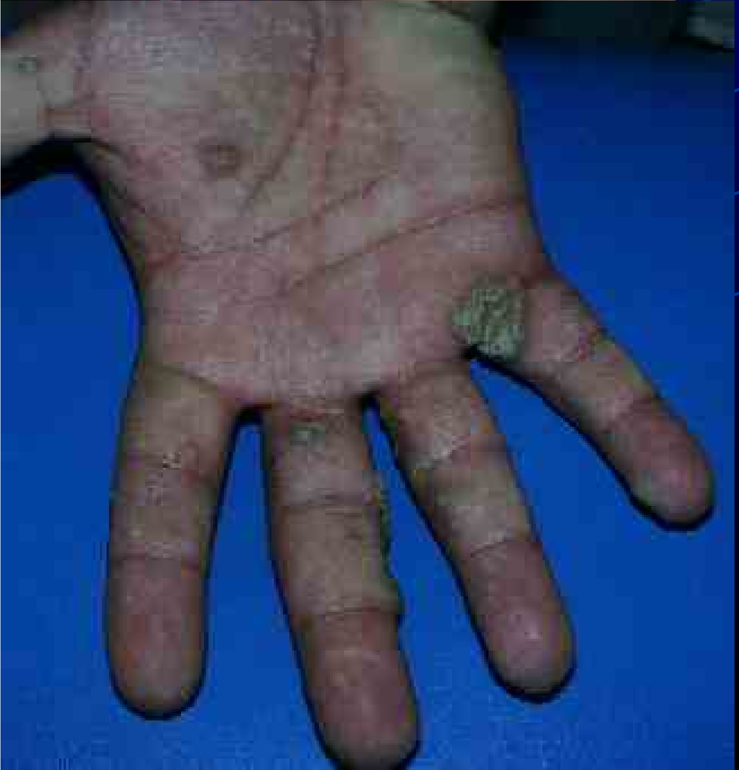


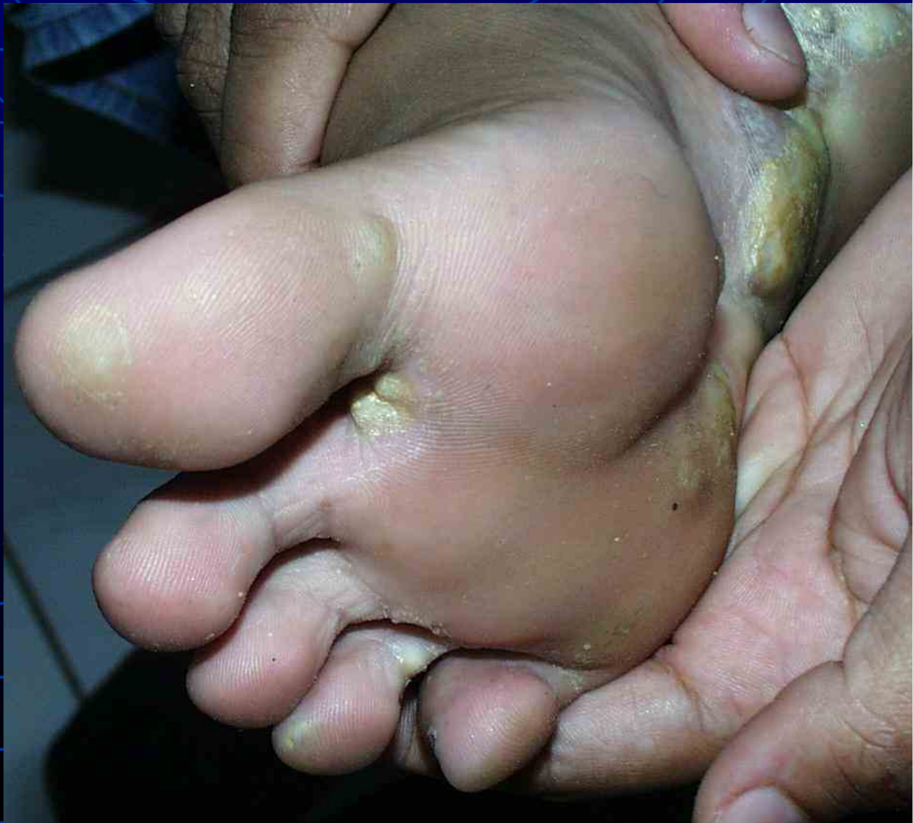


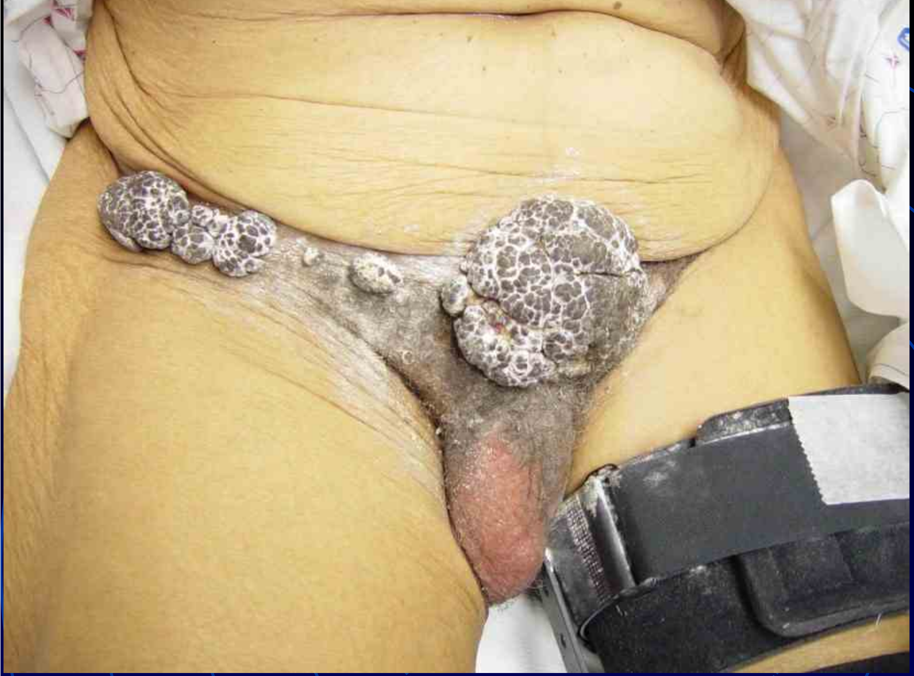
Warts

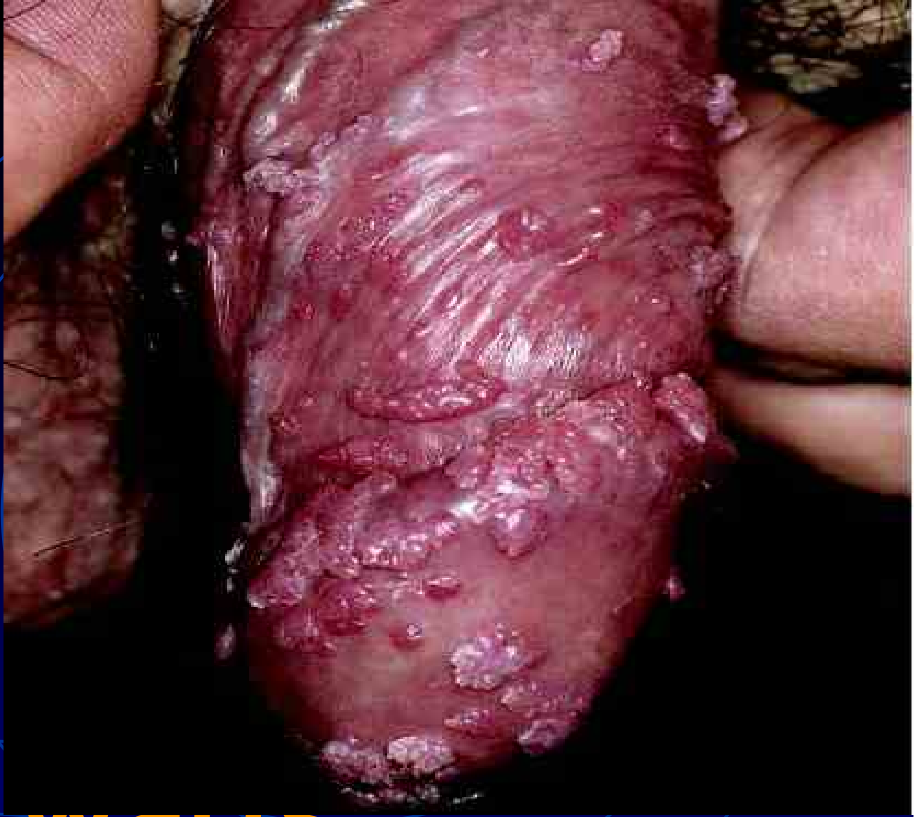



Copyright 2001, Johns Hopkins University

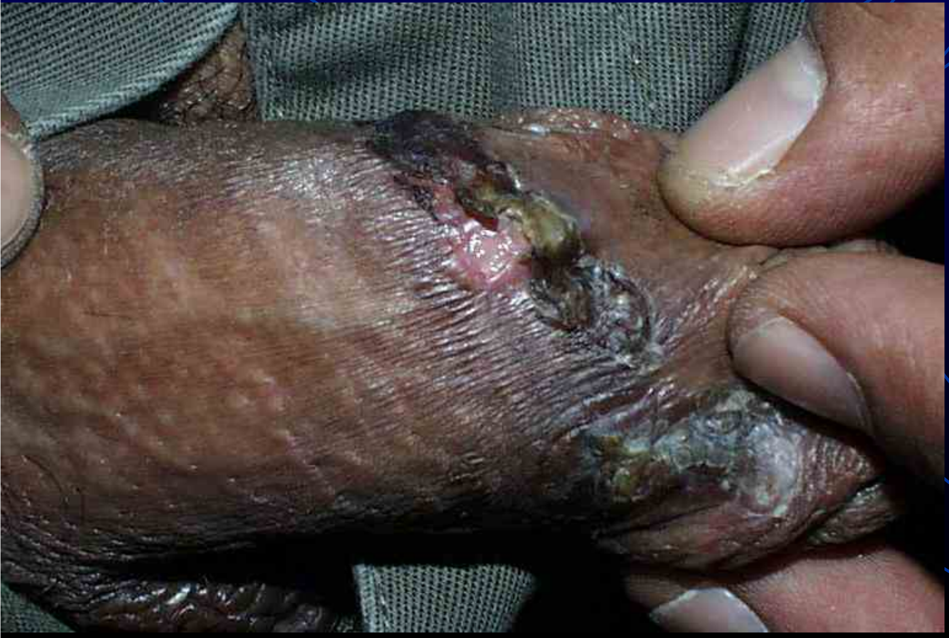


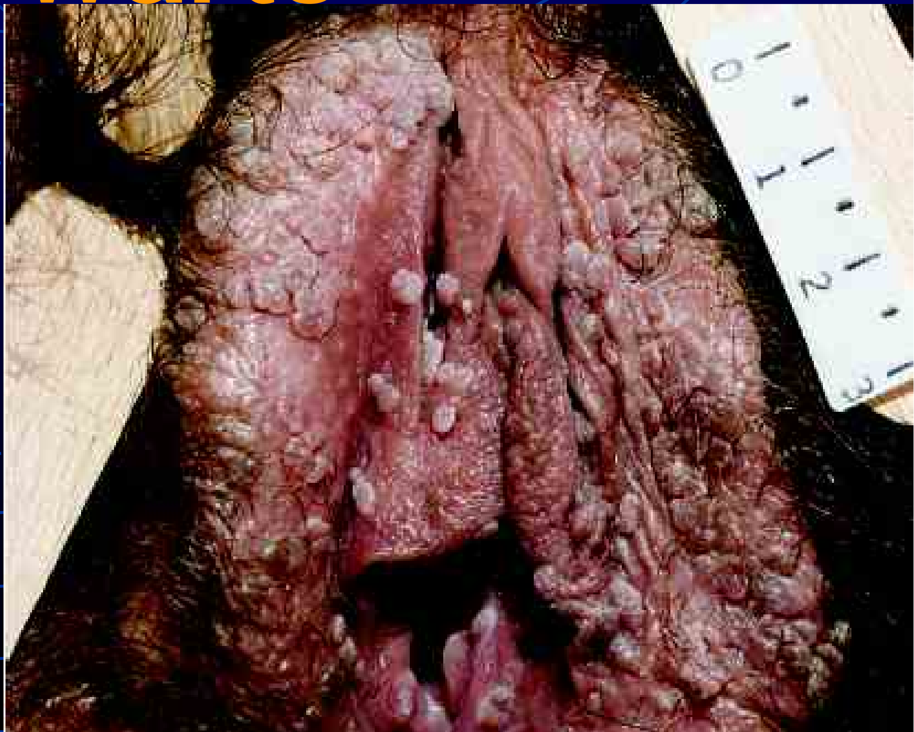






Anogenital warts





Treatment

- Chemical treatment:
    - Salicylic acid 15-20%.
    - Podophyllin 10-25% for C. acuminata.
    - Formalin 2-3% for plantar warts.
    - imoqumod (aldara) for C. acuminata.
- Electrocautery.
- Cryotherapy.
- Laser therapy.

Molluscum contagiosum

It caused by *Molluscum contagiosum virus*. Transmission of the disease is by direct contact.

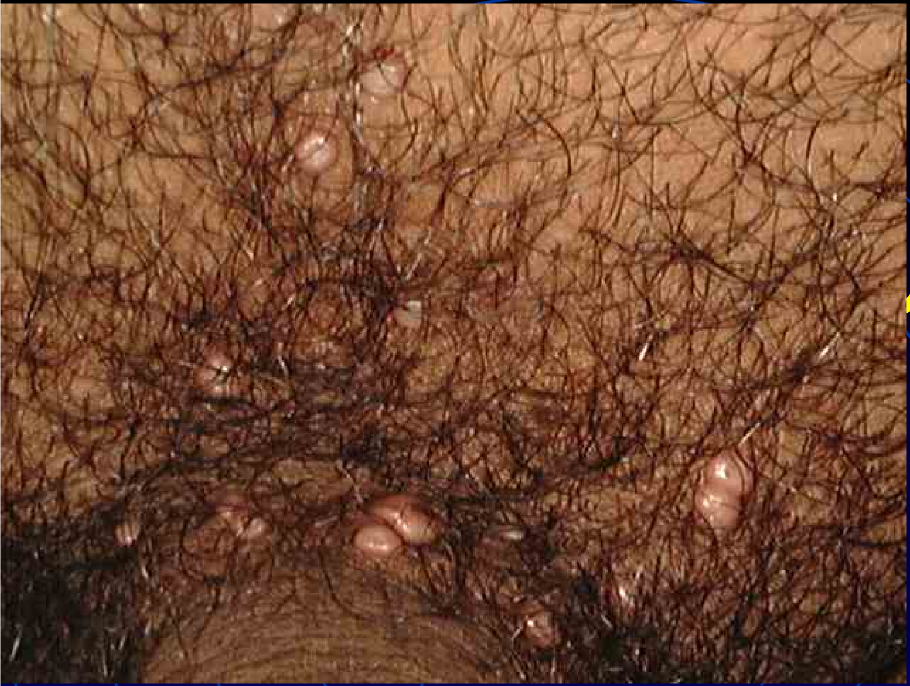
Clinical picture

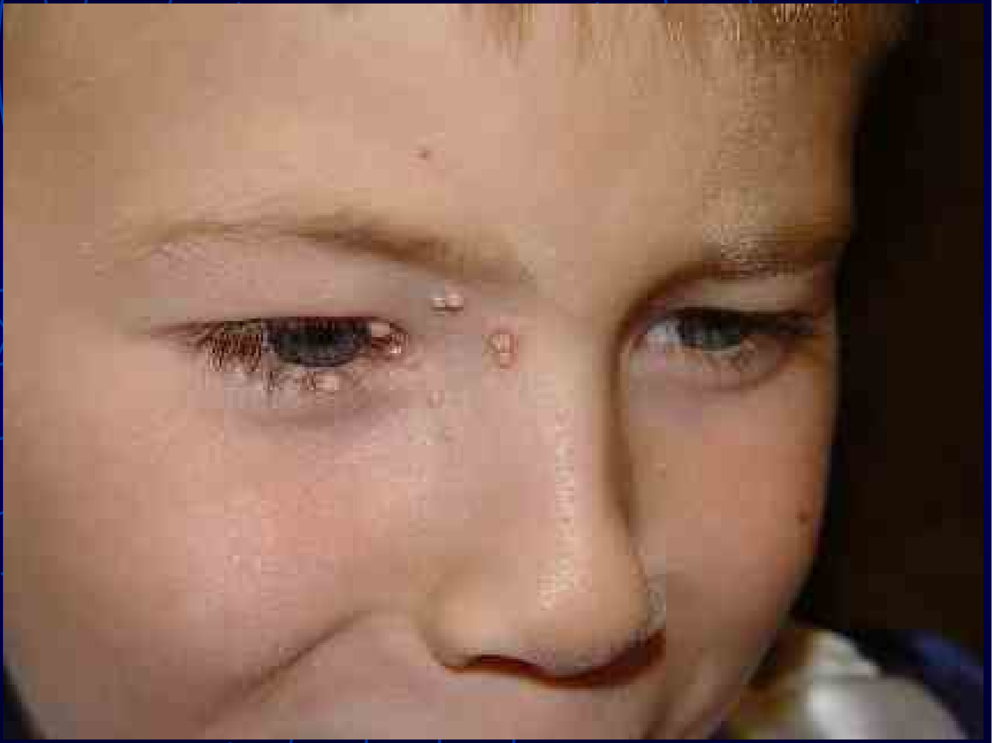
The characteristic lesion is a smooth pearly white, hemispherical, umbilicated papule, which on squeezing a white cheesy material can be expressed. It averages 3-5 mm in diameter. The lesions are usually multiple and occur mainly on the face, trunk and extremities.

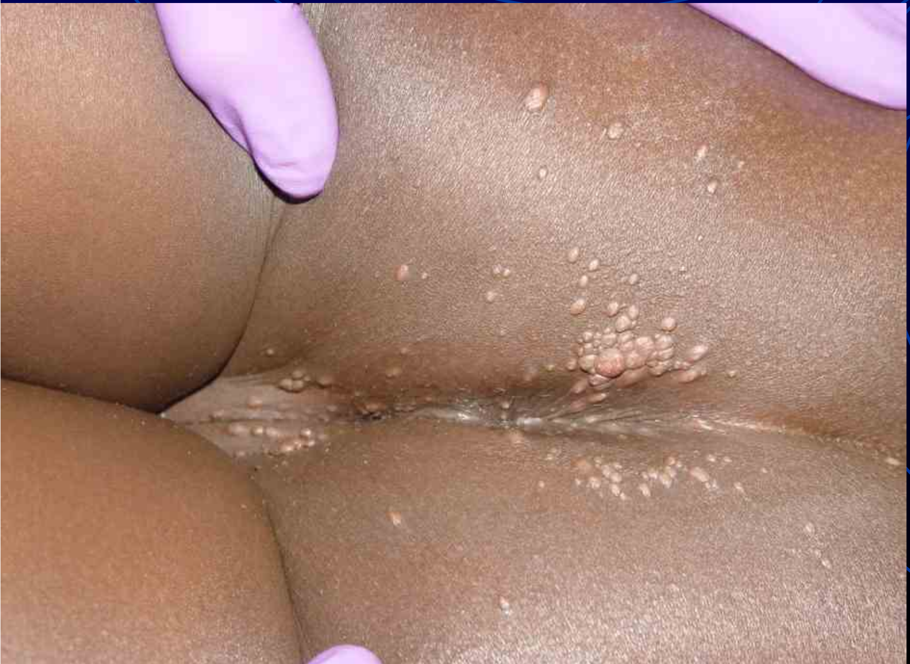
Treatment

- Spontaneous disappearance of the lesions may occur.
- Curettage and diathermy.
- Expression of the contents of the lesion by squeezing with forceps.
- Salicylic acid 15-20%.

uscum contagiosum







CUTANEOUS FUNGAL INFECTIONS

FUNGAL INFECTIONS

1. The skin constitutes the main site of recognizable fungal infections in humans, and these infections can be divided into superficial or deep (systemic) infections. Superficial infections are restricted to the skin and its appendages, while deep infections (e.g. blastomycosis or mycetoma) are usually *starting in the internal organs and involve skin secondarily.*

Fungal infections may be caused by:

1- *Dermatophytes*, these are the causative fungi of superficial infections. They are classified into 3 genera: Microsporum, Trichophyton and Epidermophyton.

2- *Yeasts* e.g. Malassezia furfur (the cause of pityriasis versicolor), and candida albicans.

Source of infection.

It may be from human (anthropophilic species), from animals e.g. cats, dogs (zoophilic species) and rarely from soil (geophilic species).

Morphology of Fungi

- **Fungi**
  - **Multicellular**
    Dermatophytes
    Have three genera:
    - **Microsporum**
      M
    - **Trichophyton**
      T
    - **Epidermophyton**
      E
  - **Dimorphic**
    Candida
    Malassezia furfur
    (pathogenic)
  - **Unicellular**
    Candida
    Pityrosporum
    (Commensal)

Clinical types of dermatophyte infections

According to the affected site, dermatophyte infections are classified into:

- Tinea capitis.
- Tinea corporis or tinea circinata.
- Tinea barbae.
- Tinea cruris.
- Tinea pedis.
- Onychomycosis (fungus infection of the nails).

Clinical Types

Ringworm infections are named according to the affected site:

1. *Scalp → tinea capitis.*
2. *Body → tinea corporis or T. circinata.*
3. *Beard & mustache → tinea barbae.*
4. *Groin → tinea cruris.*
5. *Feet → tinea pedis.*
6. *Hands → tinea manuum.*
7. *Nails → tinea unguium or Onychomycosis.*

Tinea capitis (Ringworm of the scalp)

Tinea capitis is an infectious disease occurring chiefly in school children and is more frequent in boys than girls. The main causative fungi are *Trichophyton violaceum* and *Microsporum canis and Trichophyton schoenleinii*

Tinea Capitis

Clinical Types:

1. Scaly ringworm.
2. Black-dot.
3. Kerion.
4. Favus.

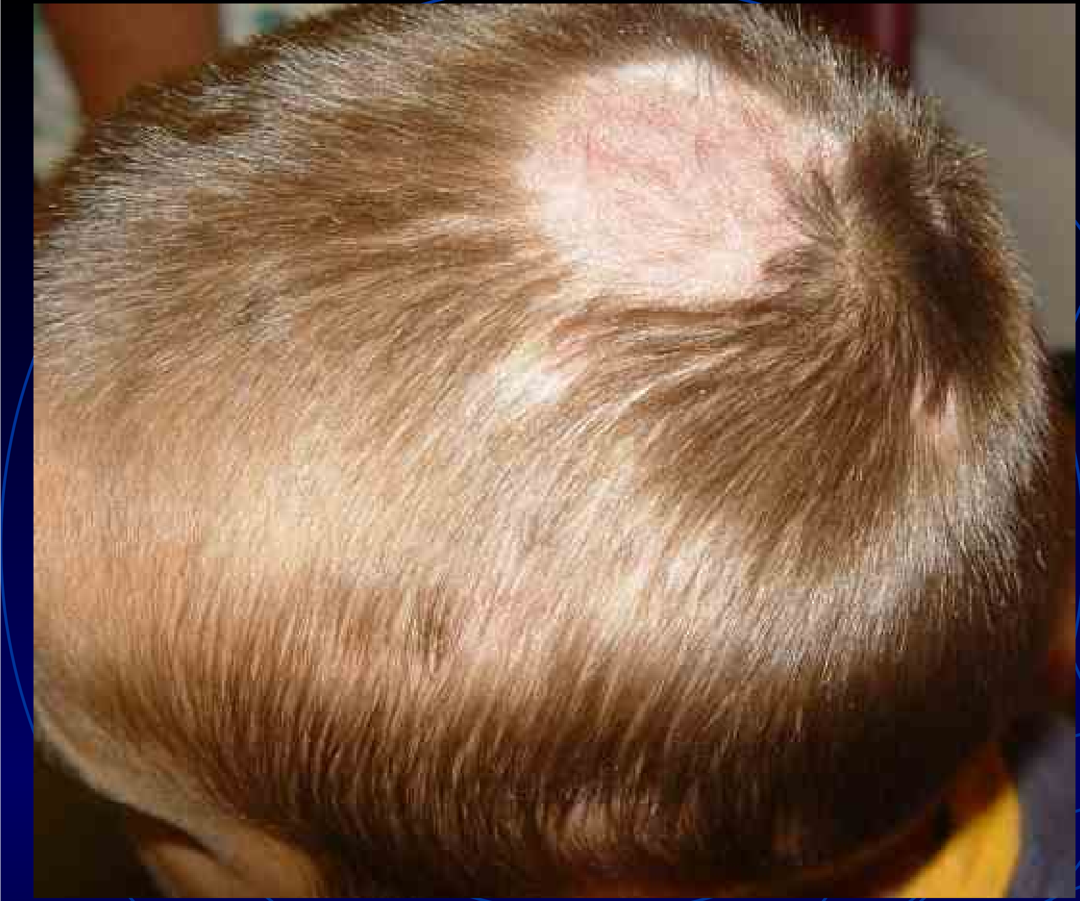
Clinical picture:

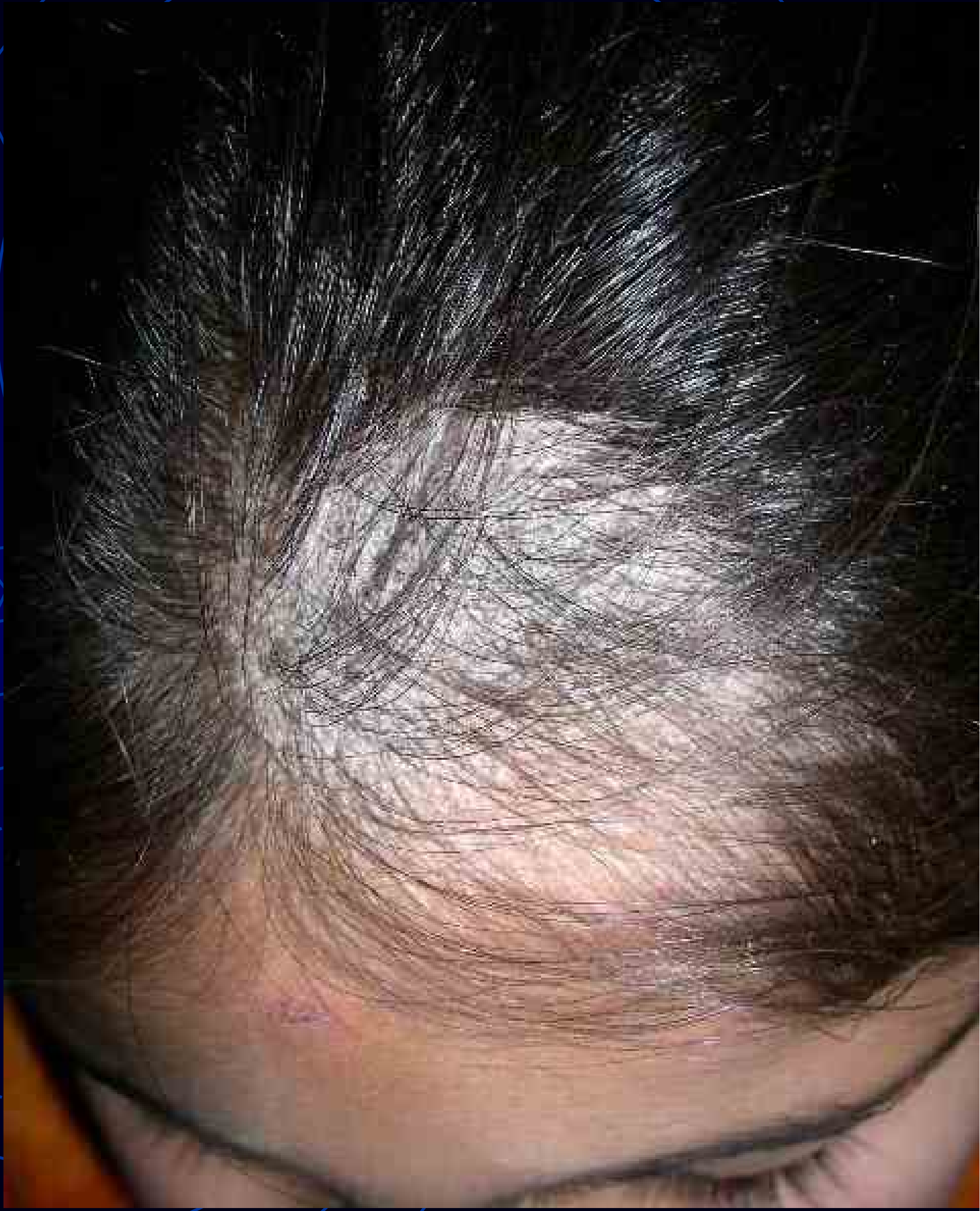
1. Scaly type.

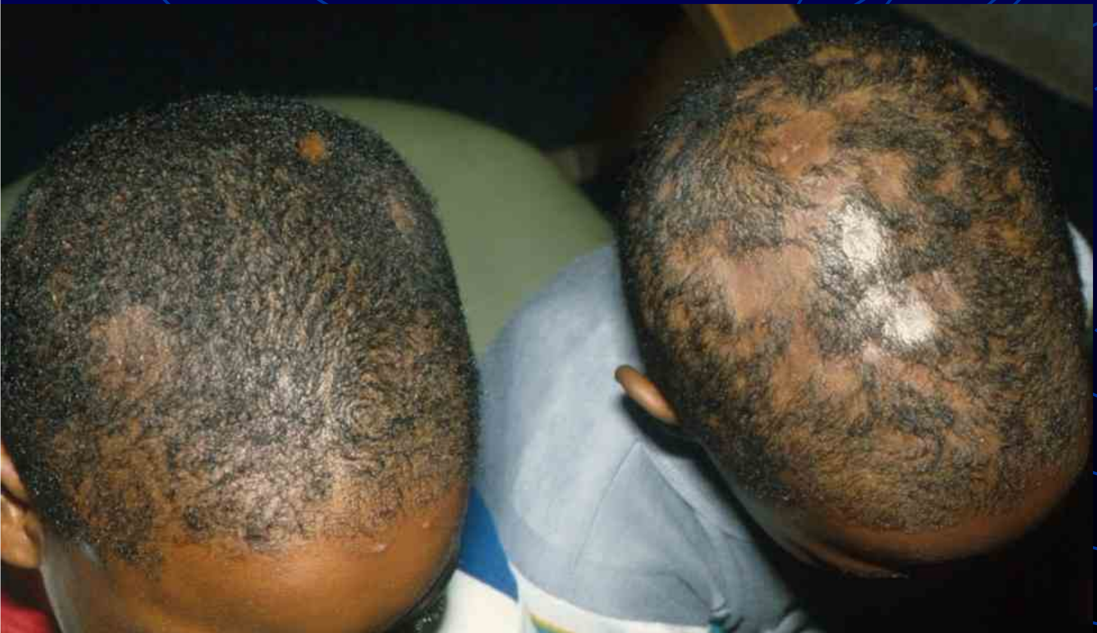
It is characterized by single or multiple scaly patches, often circular in shape, and showing numerous broken-off (2-3 mm long) dull-grey (lustreless) hairs.

Differential diagnosis. Psoriasis, seborrhoeic dermatitis, pityriasis rubra pilaris

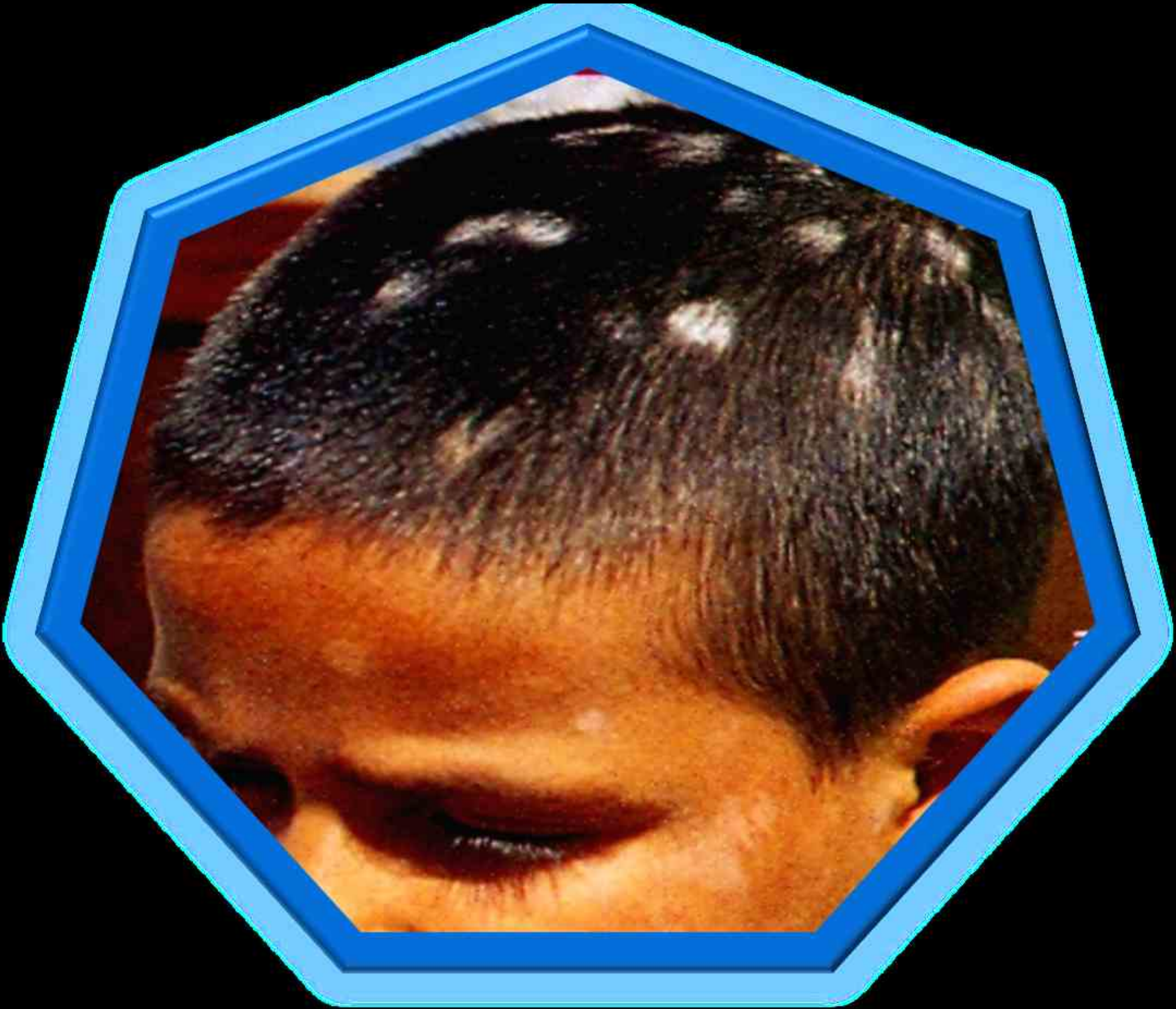
Tina capitis







Scaly Ringworm



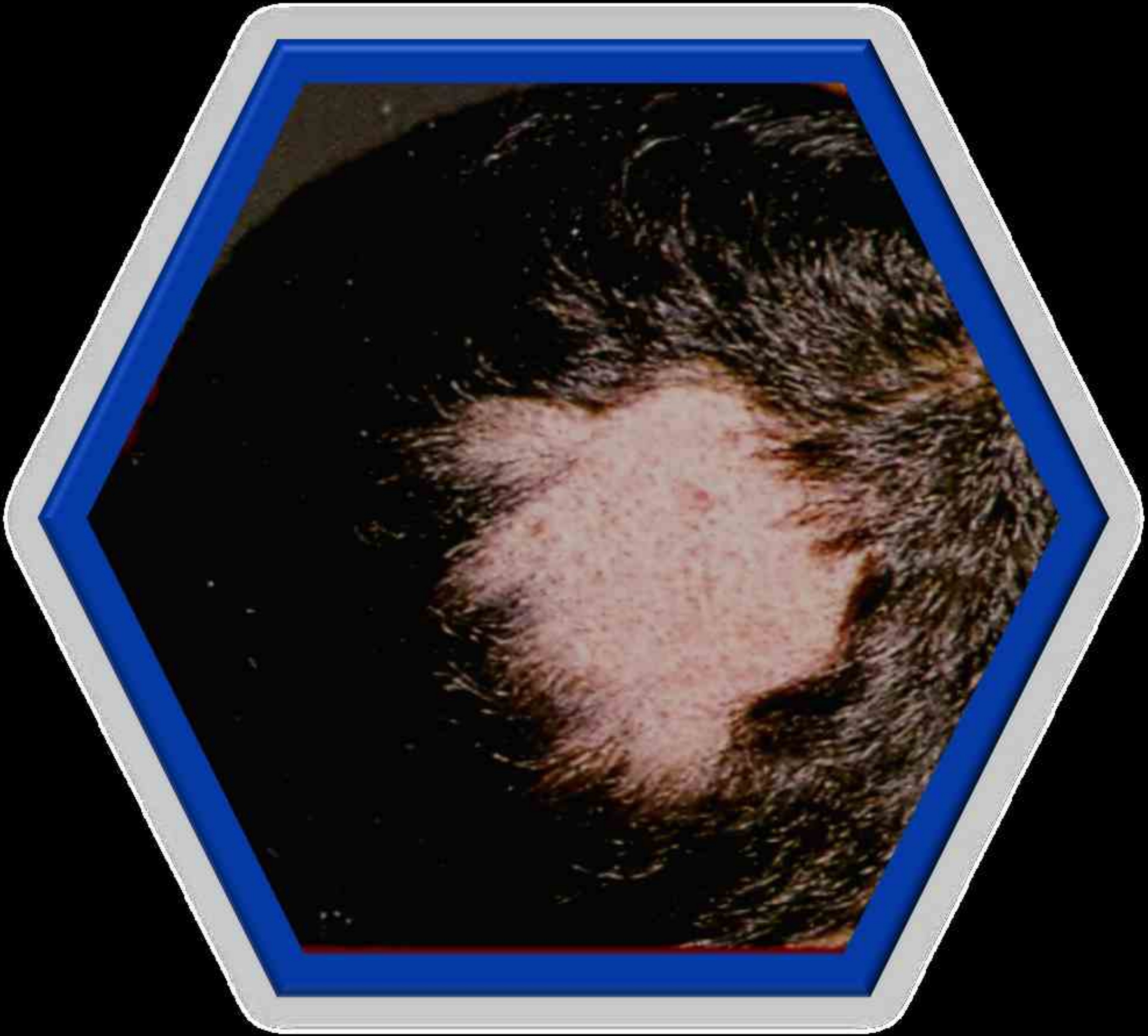
2. Black-dot type.

This type is characterized by bald patches with formation of black dots as the affected hair breaks at the surface of the scalp.

Differential diagnosis. Alopecia areata, seborrhoeic dermatitis.

In the scaly and black dot types healing usually occurs without scarring with normal growth of hairs.

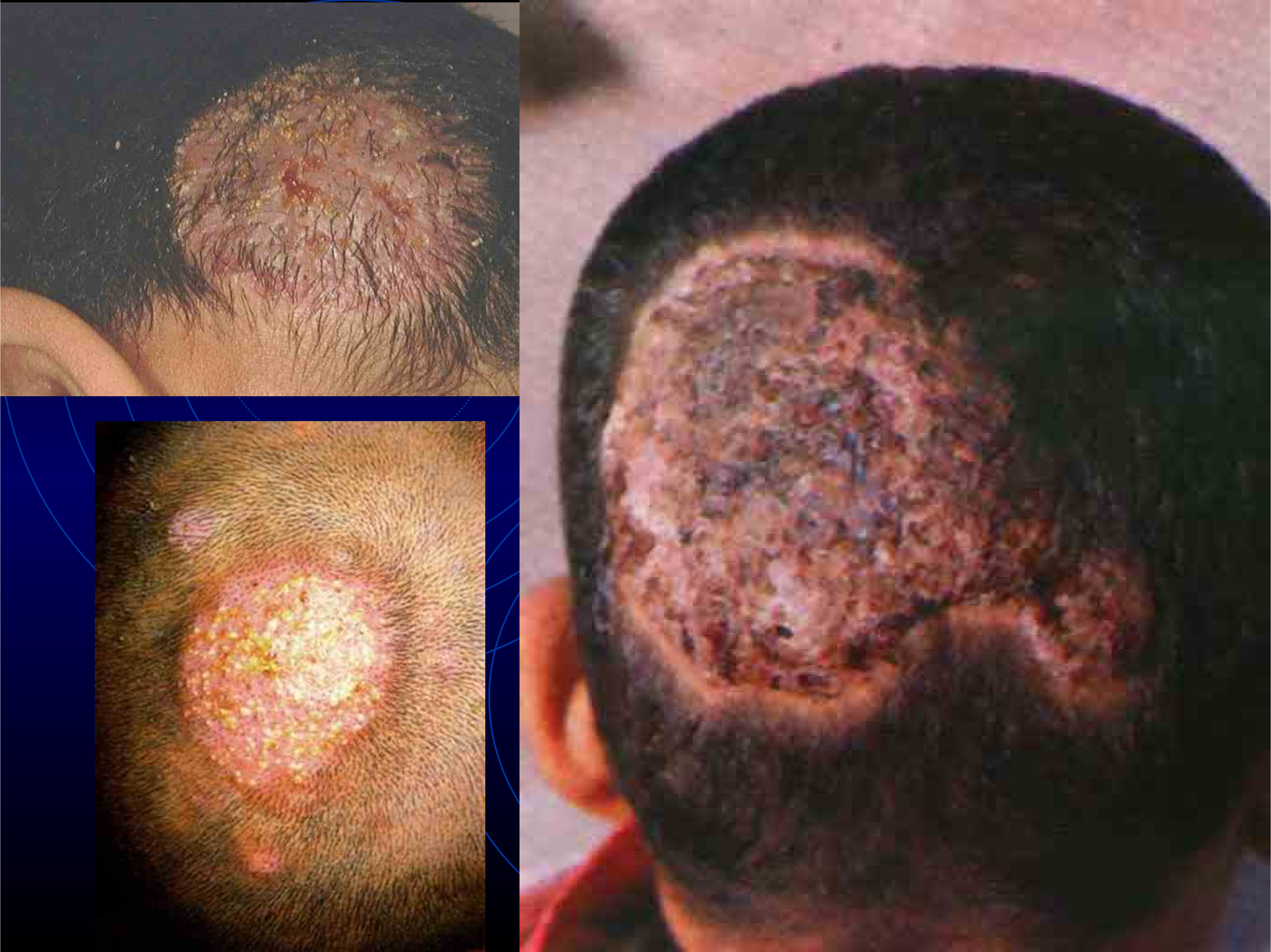
Black-dot Ringworm



3. Kerion.

This inflammatory type is usually caused by animal types of fungi and presented as boggy indurated swellings with crusting and loose hairs. Follicles may be seen discharging pus. In extensive lesions, fever, pain, and regional lymphadenopathy may be present. Kerion may be followed by scarring and permanent alopecia.

Differential diagnosis. Pyogenic abscess, impetigo



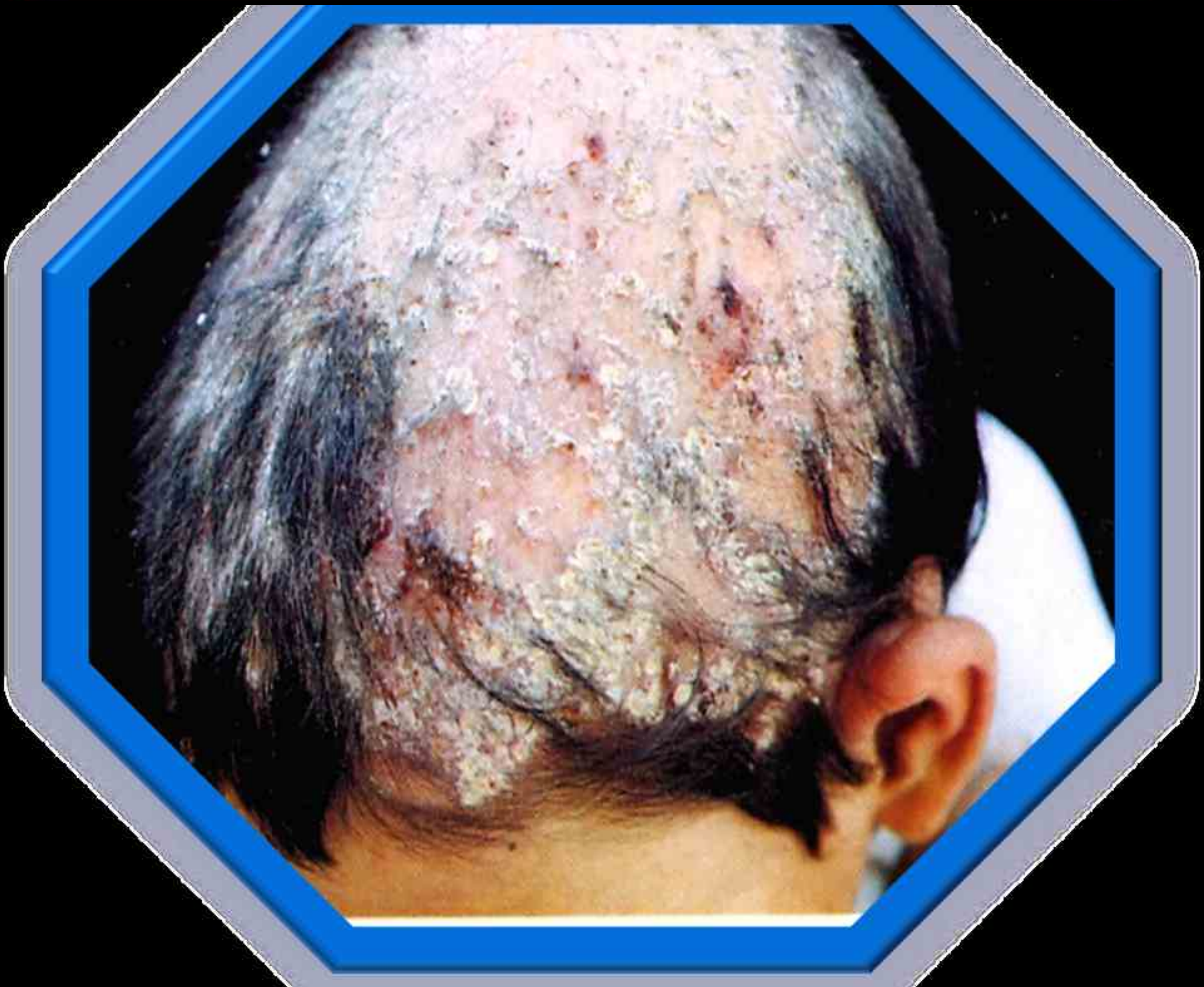
4. Favus.

It is caused by *Trichophyton schoenleinii* and is characterized by the presence of yellowish, cup-shaped crusts known as scutula . Each scutulum develops round a hair, which pierces it centrally. The scutula have a distinctive mousy odour. Cicatricial alopecia is usually found in long-standing cases.

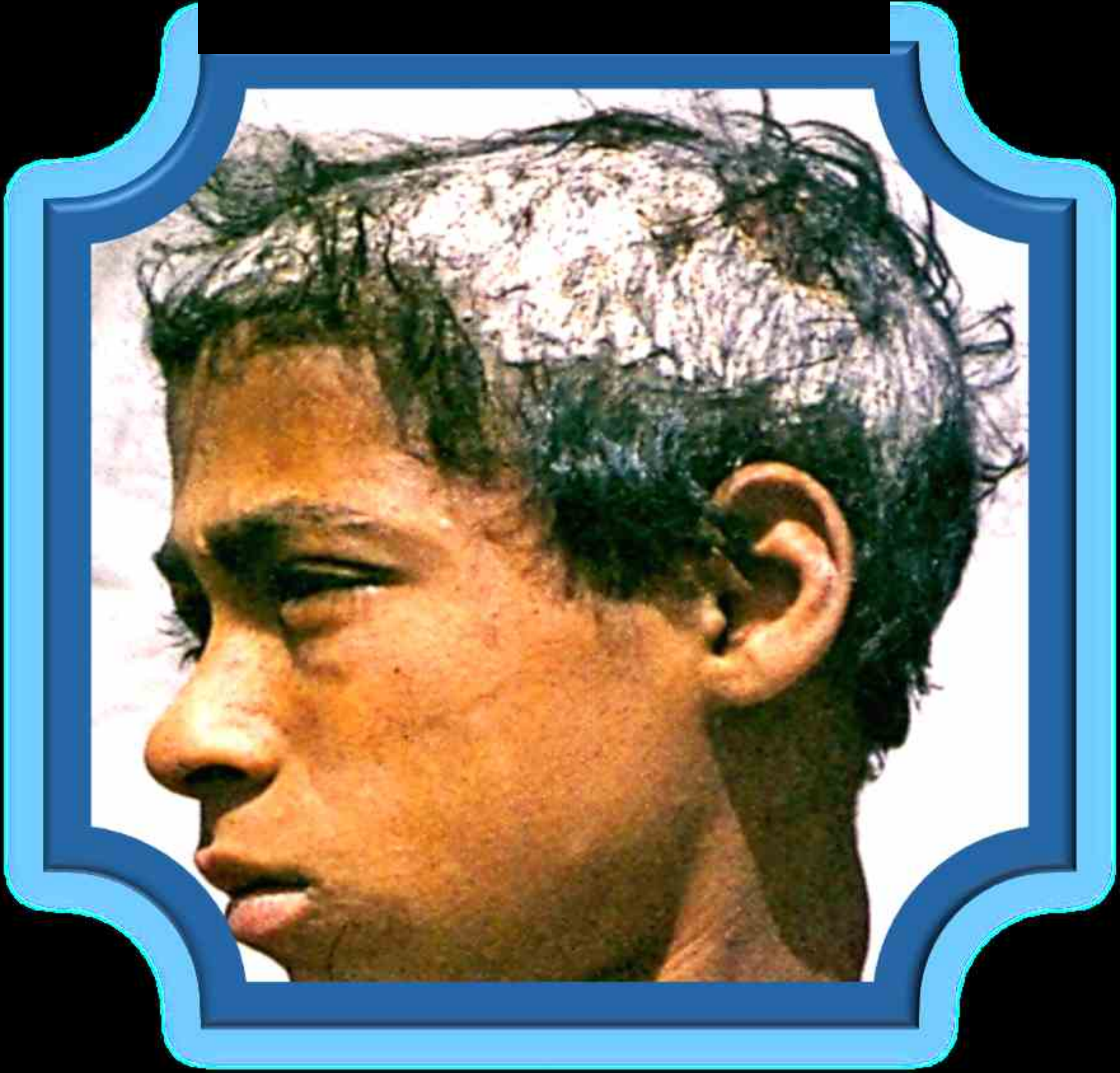
Differential diagnosis. Psoriasis, seborrhoeic dermatitis (early stages), DLE, lichen planus (atrophic stage).

Favus

(Cup-shape scutula and cicatricial alopecia)

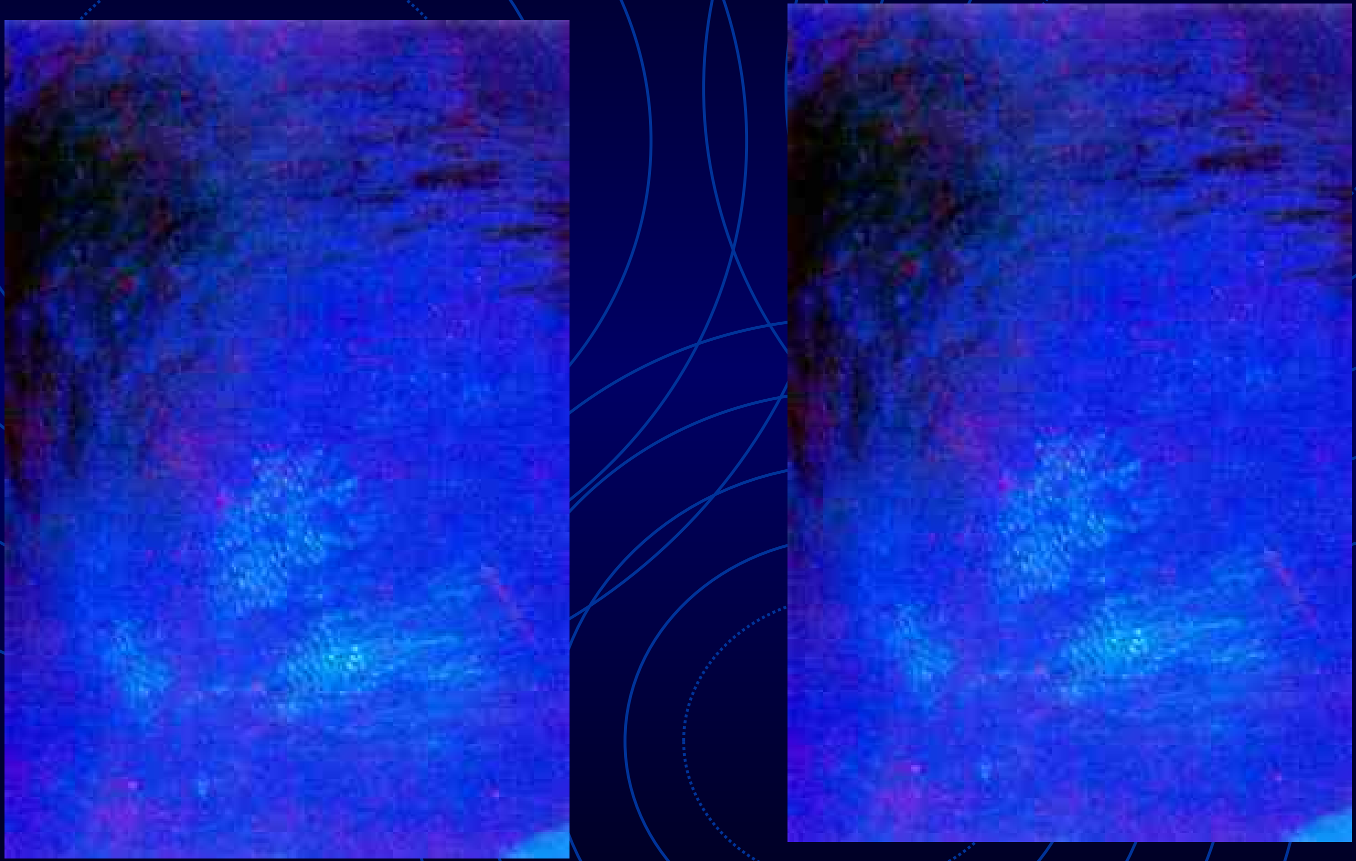


Favus



- **Modes of infection**
  - Direct contact with infected child.
  - Indirect through the use of patient’s fomities e.g. brushes and caps.
- **Diagnosis**
  - Clinical picture.
  - Wood’s light.
  - Direct microscopic examination of the infected hair and scales using 10-20% potassium hydroxide (KOH) to demonstrate the fungal spores.
  - Culture on Sabouraud’s medium.

Wood lamp examination of a gray-patch area on the scalp. In Microsporum canis infection, scalp hairs emit a diagnostic brilliant green fluorescence. Trichophyton tonsurans does not fluoresce with Wood lamp.



Treatment

A- Topical treatment

- Topical therapy has little place in the management of tinea capitis except as an adjunct to oral therapy. The main topical preparations are:
- Whitfield's ointment.
- Imidazoles, e.g. clotrimazole, econazole, ketoconazole.
- Allylamines, e.g. terbinafine.

B- Systemic treatment

- Griseofulvin, 10 mg/kg/day for at least 6 weeks and 8 weeks in favus.
- Itraconazole, fluconazole and terbinafine are used only in selected cases.

Tinea corporis (Tinea circinata)

- Tinea corporis is the ringworm infection of the glabrous skin. It commonly involves the exposed skin. The disease is more common in children.
- The lesions are characteristically circular, sharply circumscribed, erythematous and scaly. The edge of the lesion is usually elevated and more inflamed than the center (raised active edge).
- Progressive central clearing produces annular lesions.

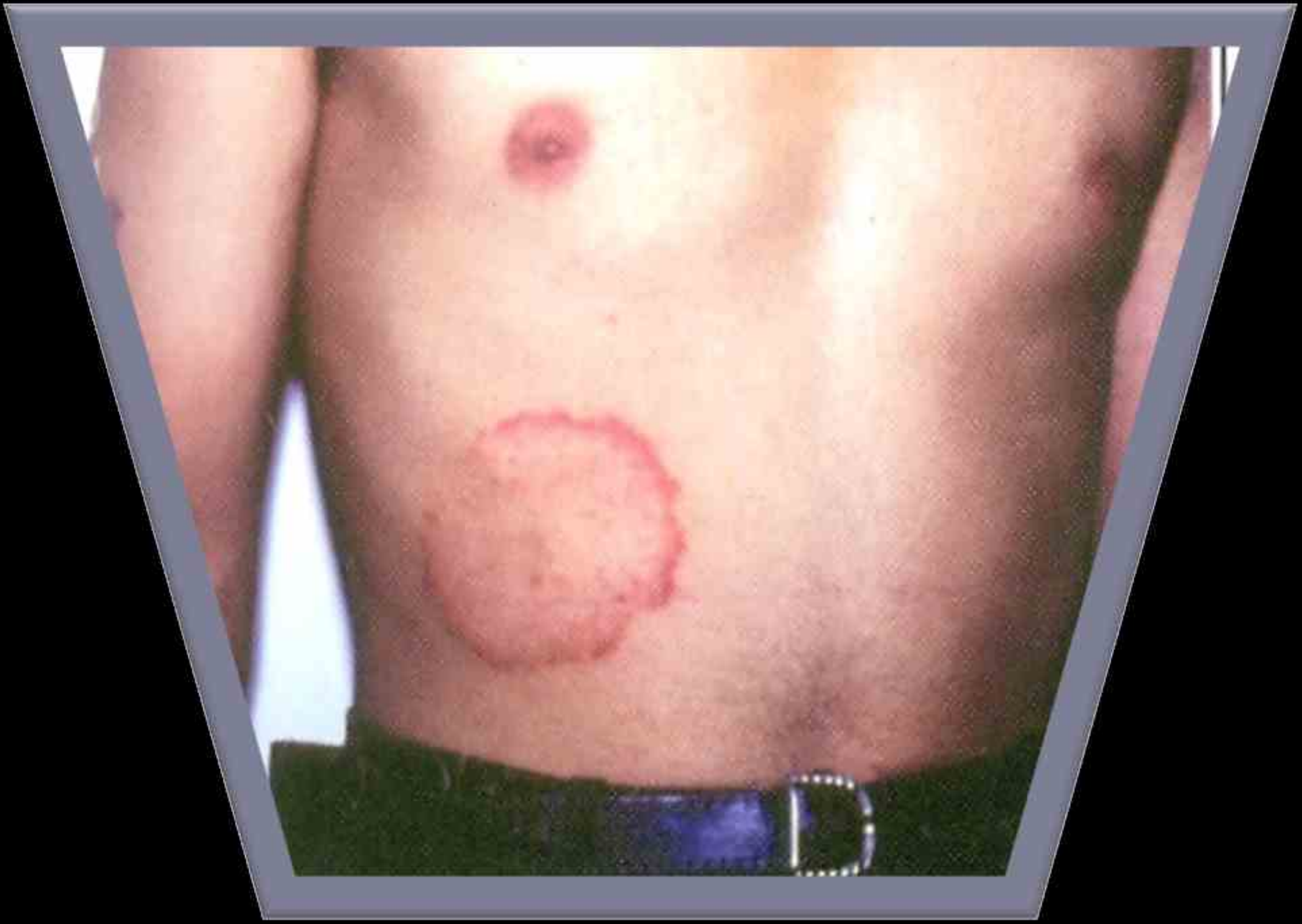
Differential diagnosis. Pityriasis rosea, circinate impetigo and discoid eczema.

Treatment

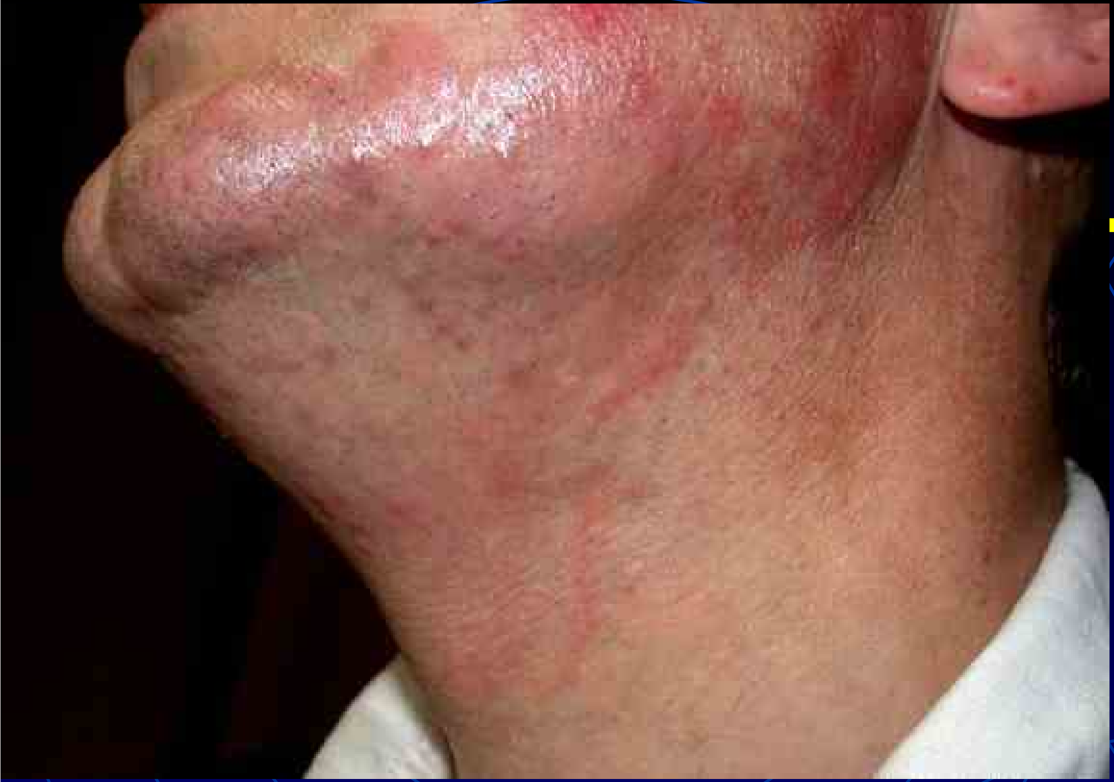
When one or two patches occur, topical treatment for 2-4 weeks is sufficient but extensive disease may require systemic antifungals.

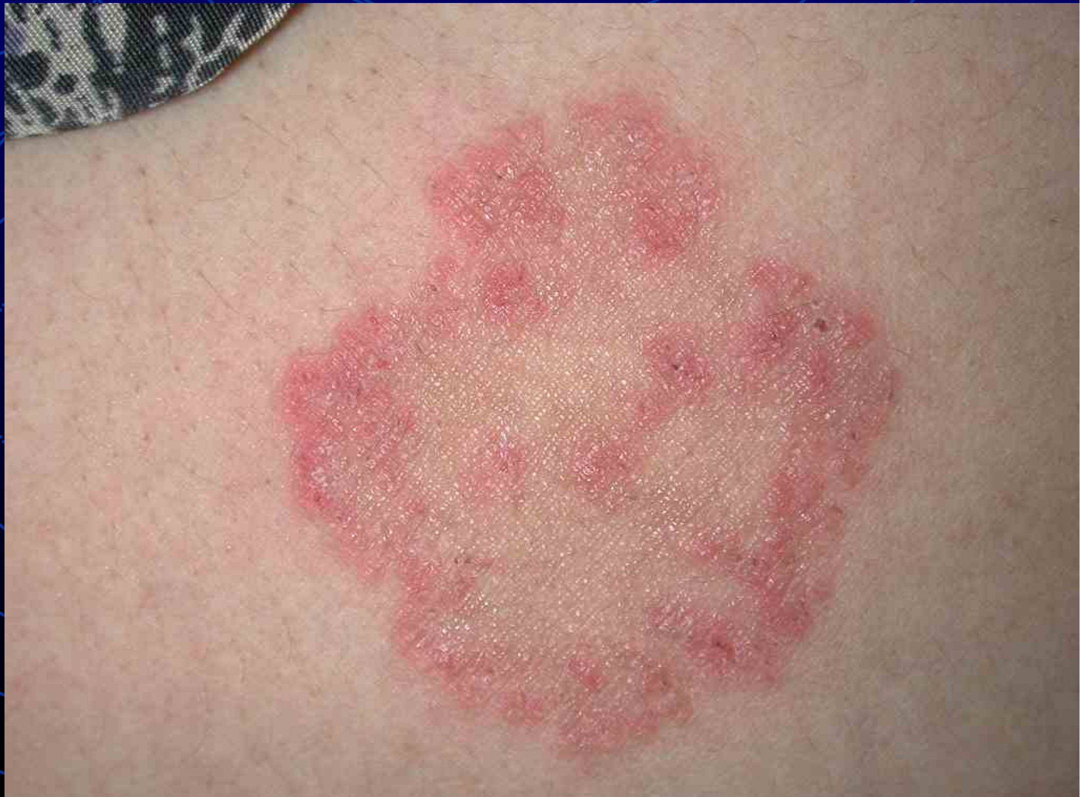
T. Circinata

Single large Patch



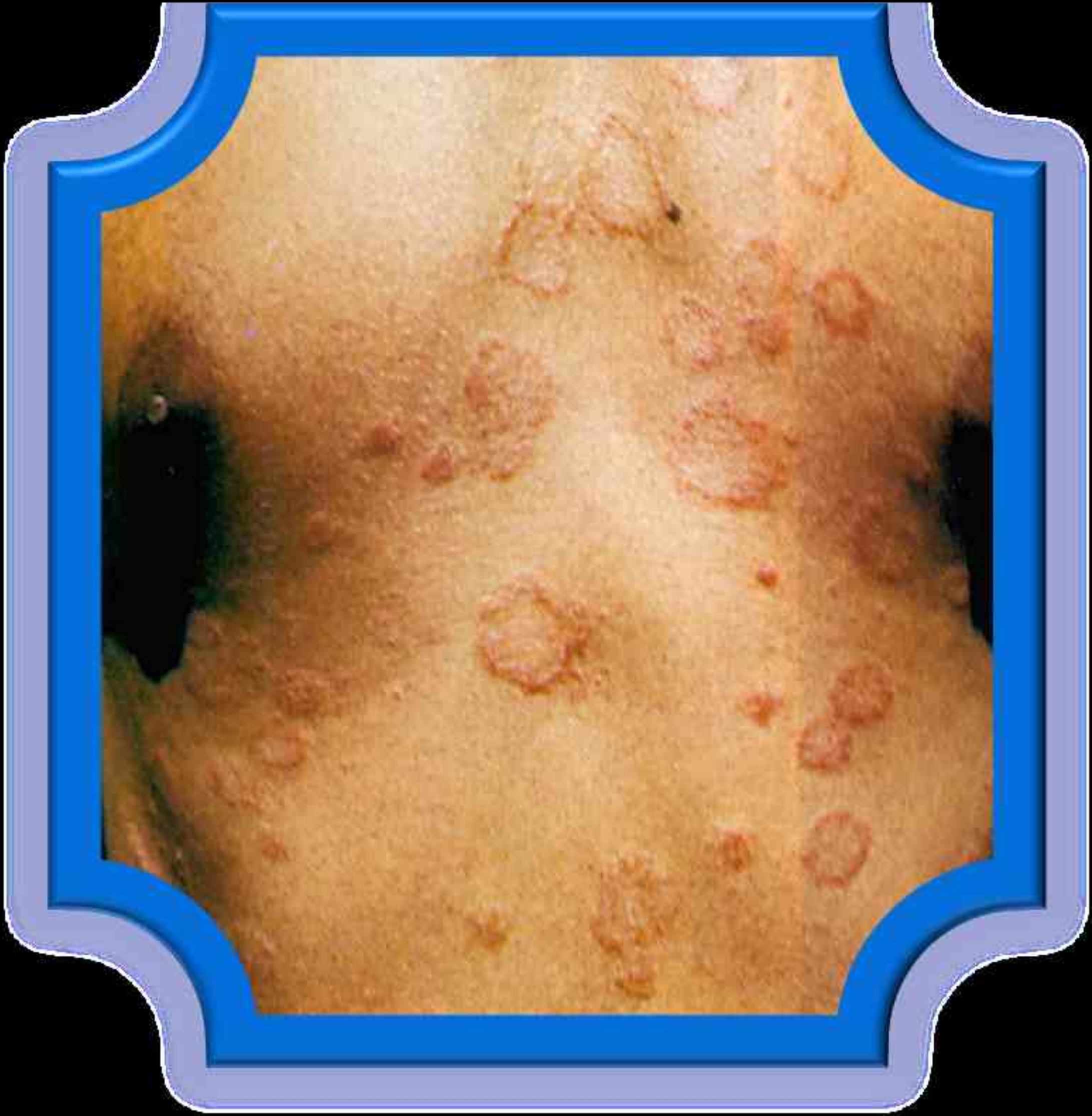
Tinea circinata





Pityriasis Rosea

D D of Tinea Circinata



Tinea barbae

- *Ringworm of the beard and mustache area.*
- Tinea barbae is a disease of adults especially those in contact with farm animals. The lesion may be presented as kerion or as tinea circinata and mostly unilateral .

Treatment

- Like tinea capitis, oral and topical antifungals are required to cure tinea

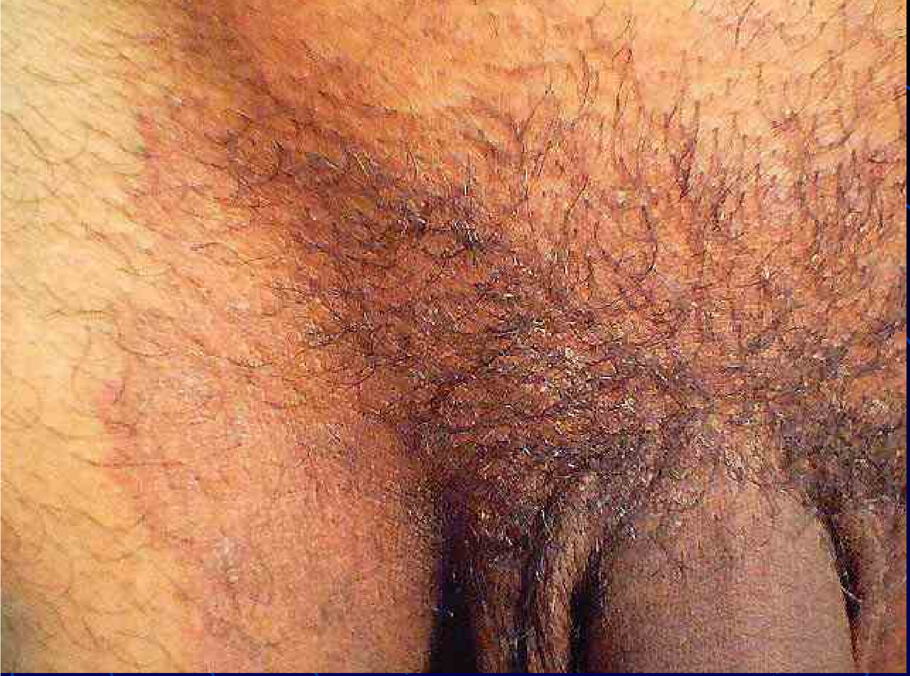
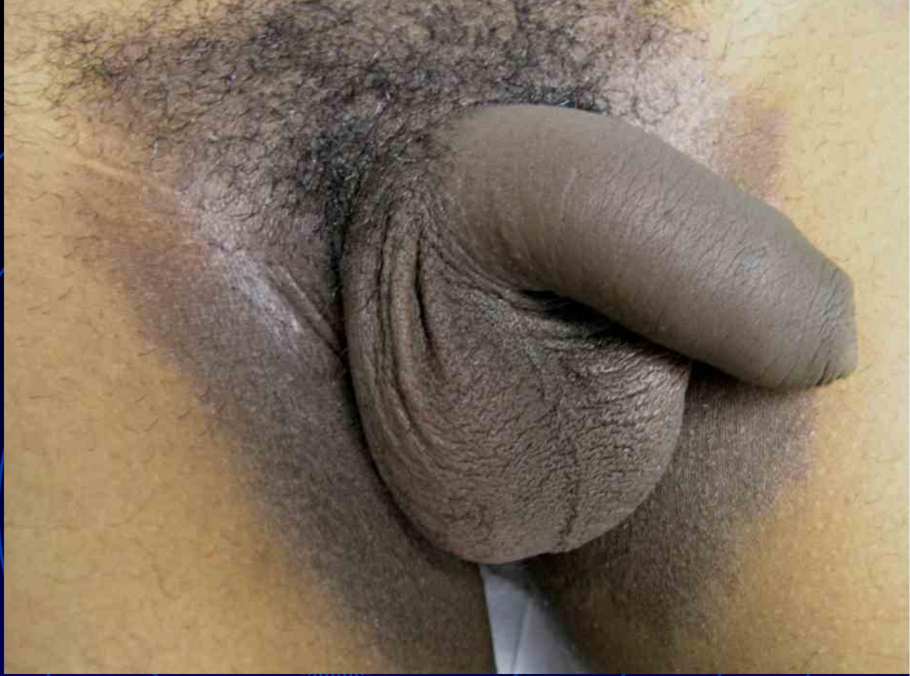
Tinea cruris

- Tinea cruris occurs mostly in men on the upper and inner surfaces of the thighs, especially during the hot summer months. It begins as a small erythematous and scaling patch that spreads peripherally and partly clears in the center. The edge of the lesion is usually well defined and may have vesicles, pustules or papules. Itching is common in tinea cruris.

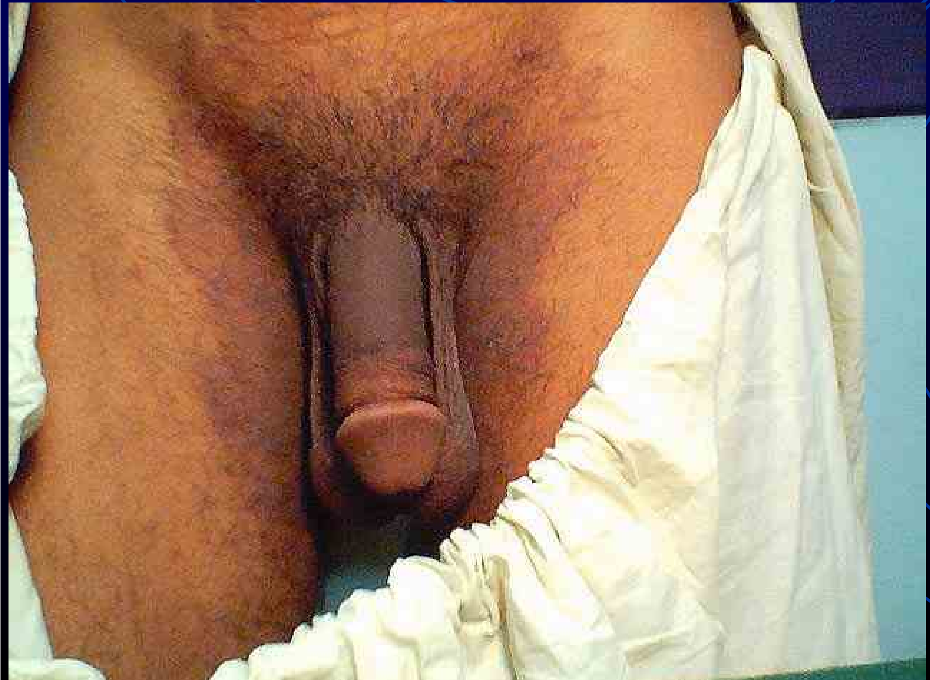
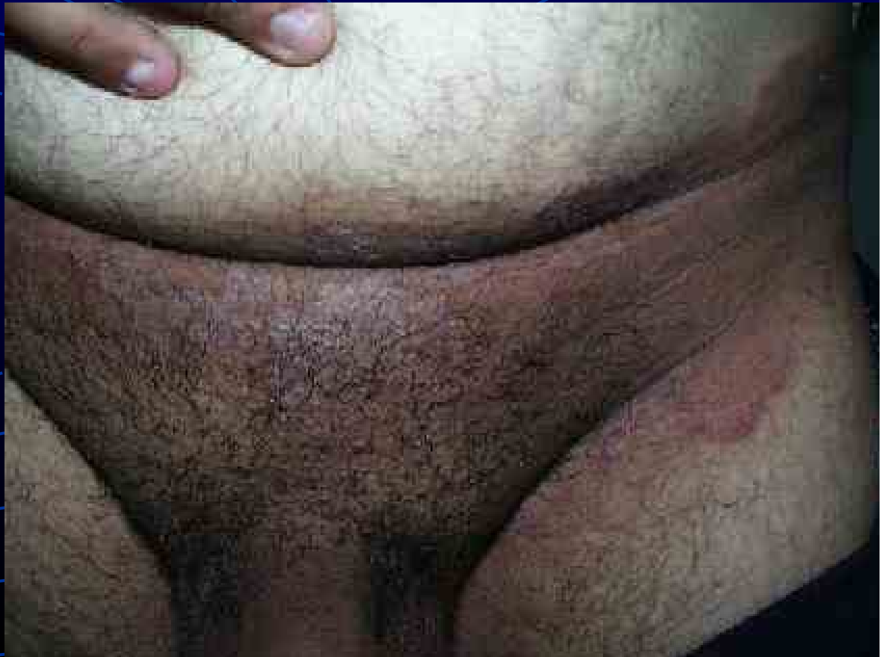
Differential diagnosis. Erythrasma, seborrhoeic dermatitis, flexural psoriasis and candidal intertrigo.

Treatment

- The area should be kept as dry as possible by the wearing of loose underclothing, antifungal powders are also helpful.
- Specific topical and oral antifungals as those for tinea corporis.

Tinea cruris

Tinea pedis

- It is also called athlete's foot. It is more common in adults, and more in males. The wearing of shoes, communal showers and swimming baths, and hyperhidrosis are important predisposing factors. T. pedis is due to *Trichophyton (T.) rubrum* ,*T. interdigitale*, previously called *T. mentagrophytes var. interdigitale* ,*Epidermophyton floccosum*

Clinical varieties

1- Interdigital variety. It is the commonest form of tinea pedis and characterized by peeling, maceration and fissuring affecting the lateral toe clefts, and sometimes spreading to involve the undersurface of the toes.

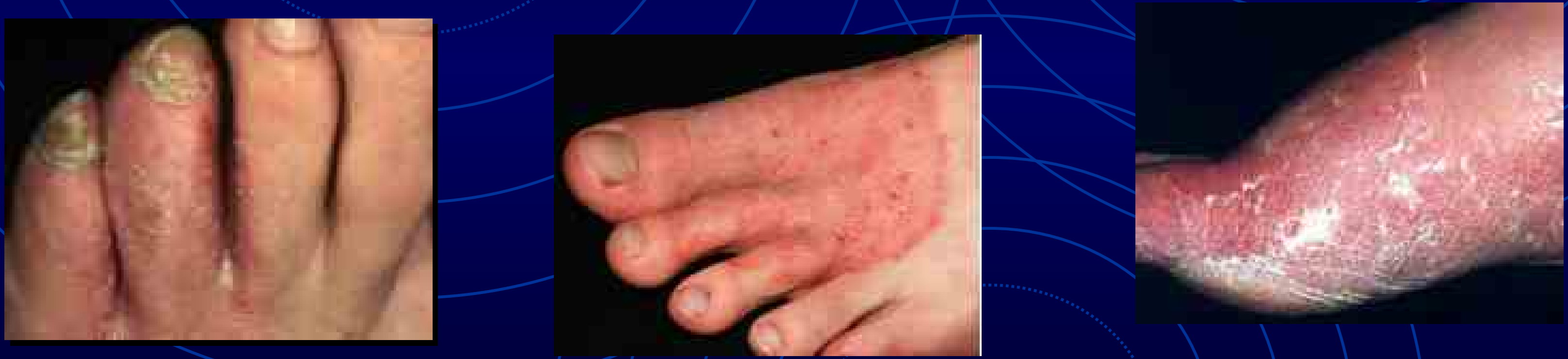
2- Squamous hyperkeratotic variety. This variety is very chronic and resistant to treatment and characterized by erythema and scaling that may involve the entire sole and sides of the foot.

3- Vesiculobullous variety. It is characterized by acute vesicular

In tinea pedis, the patient may complain of itching, bad smell, and secondary bacterial infection.

Treatment

- Dry the feet thoroughly after bathing.
- Antifungal powder on the feet for susceptible persons.
- Topical antifungals for mild cases.
- Systemic antifungals for extensive and chronic cases.



Onychomycosis (Tinea unguium)

- In onychomycosis, the nailplate becomes thickened, discolored, and cracked with accumulation of subungual hyperkeratosis. A later phase of invasion may lead to onycholysis and massive destruction of the nailplate.

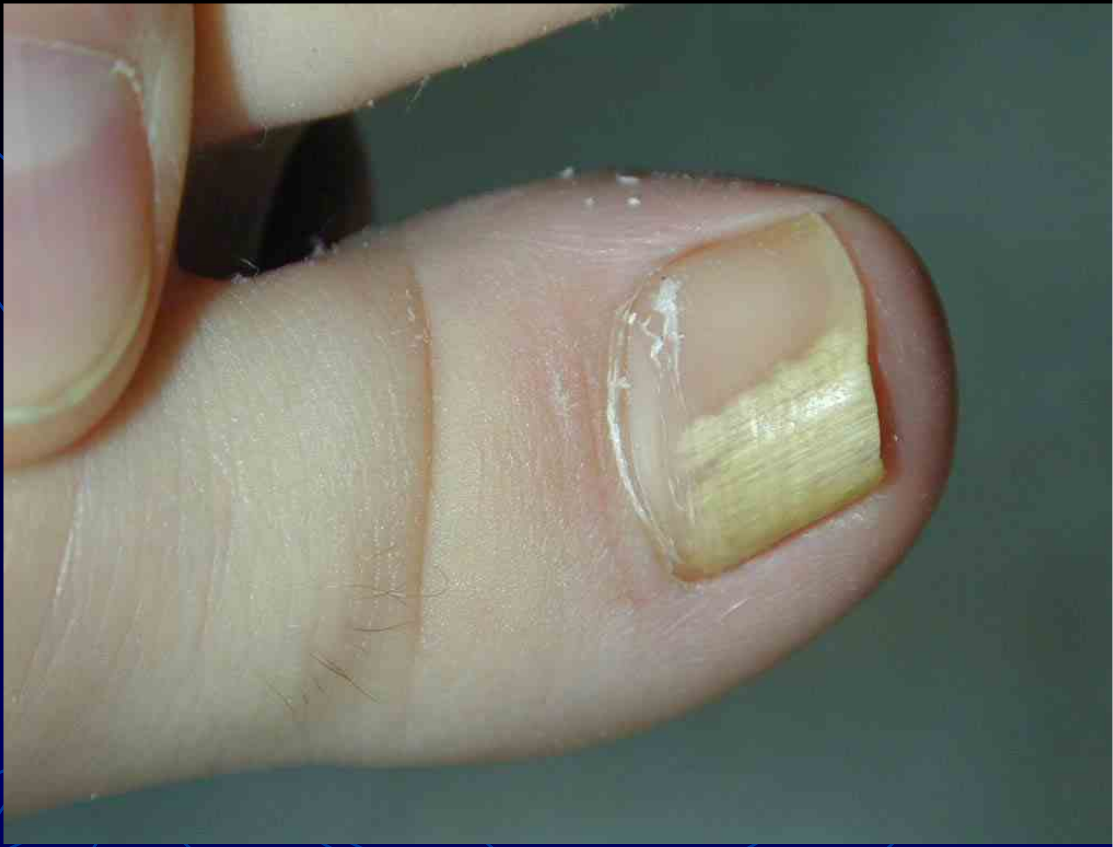
❖ *Clinically the nails may show :*

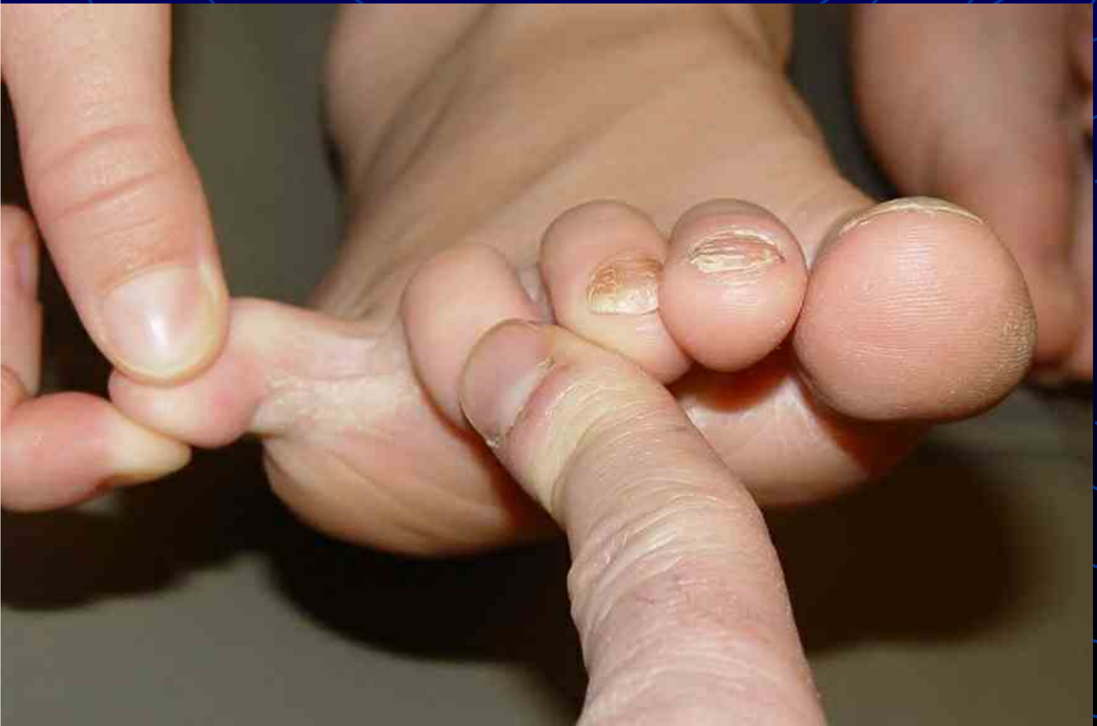
  - *Discoloration.*
  - *Thickening.*
  - *Cracking.*
  - *Subungular hyperkeratosis.*
  - *Onycholysis (separation of nail from its base).*

Treatment

- Onychomycosis is generally treated by oral antifungals.

Onychomycosis





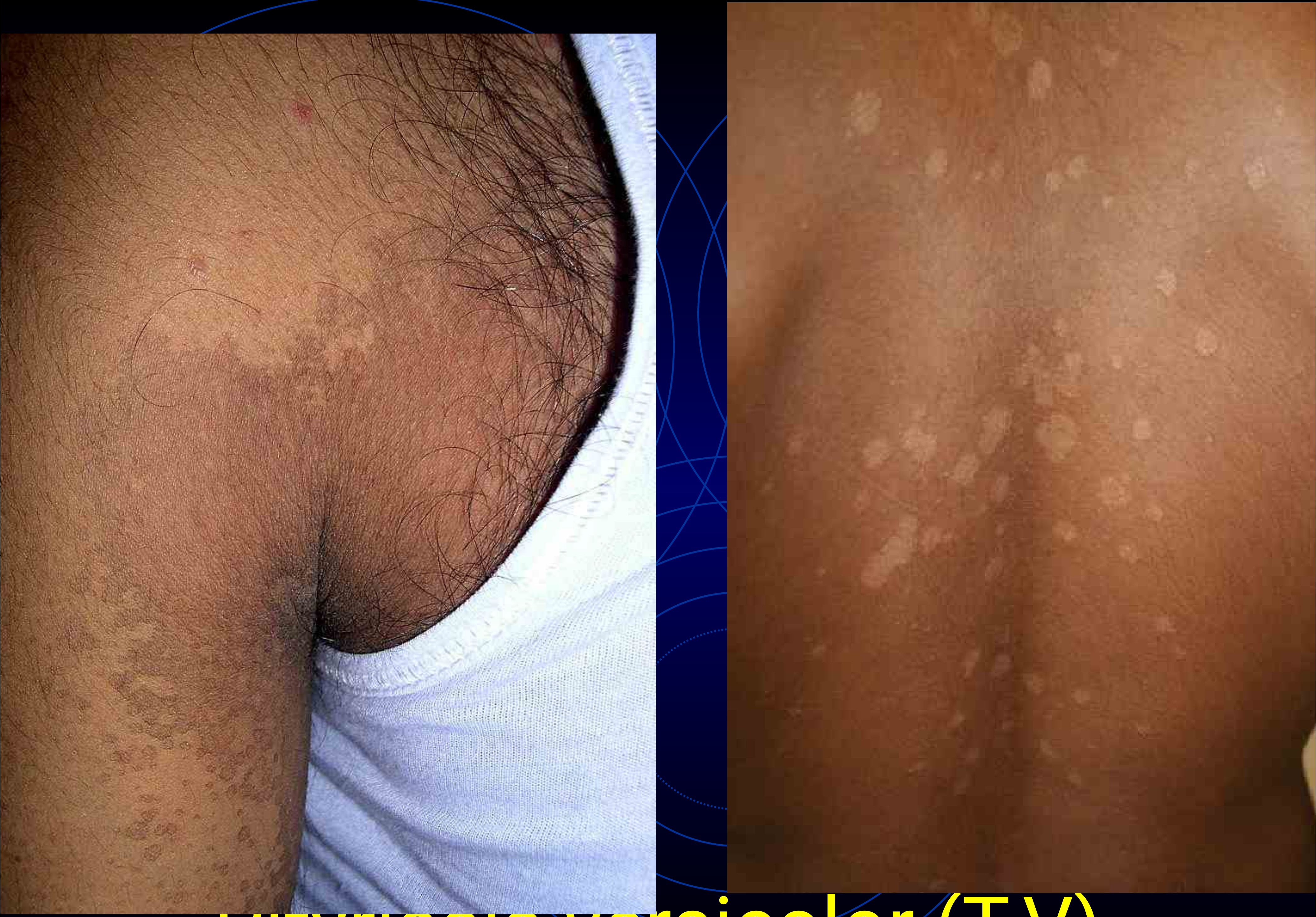
Pityriasis versicolor (T.V)

Pityriasis versicolor is a chronic fungal infection of the skin caused by the yeast *Malassezia furfur* which is one of the normal flora of the skin. The onset is more often in the warmer months of the year. The disease affects mainly young adults.

Clinical picture

The patient usually complains only of a patchy and varying change of skin color, but mild irritation is sometimes noticed. The primary lesion is a sharply demarcated macule covered by fine branny scales. The lesions may coalesce to form large confluent areas, and scattered oval patches. The patches are yellowish or brownish in white skin and hypopigmented in dark skin.

The sites of predilection are the chest, the upper arms, the abdomen, back, neck, and intertriginous areas. Relapse of the disease is very common.



Pityriasis versicolor (T.V)

Diagnosis of PV

1. *Clinical picture.*
2. *Wood's light examination → yellow-brown fluorescence.*
3. *Light microscopy of skin scrap from scales + KOH → mycelia and spores (spaghetti and meat-balls appearance).*
4. *Culture: not necessary for diagnosis, but can done on Sabouraud's or special media →Yeast-like cells (only) with rare mycelia.*

DD:

1. *Vitiligo.*
2. *Pityriasis rosea.*
3. *Seborrheic dermatitis.*
4. *Melasma.*
5. *Pityriasis alba.*
6. *Secondary syphilis.*

Treatment

- Topical antifungals daily for 2-3 weeks.
- Ketoconazole 2% shampoo.
- Selenium sulphide 2-5% shampoo.
- Sodium hyposulphite 20% solution.
- Oral ketoconazole, fluconazole and itraconazole are very effective but used only in selected cases.
- Oral terbinafine and griseofulvin are ineffective in the treatment of tinea versicolor.

Candidiasis

- Candidiasis is an infection caused by the yeast *candida albicans*, or occasionally by other species of candida. *Candida albicans* may be a normal inhibitant at various sites (gut, mouth and vagina) and able to behave as a pathogen only in the presence of impaired immune response, or in body folds (inframammary, axillary, inguinal, or vulvovaginal).

- **Predisposing factors**
- Warmth, moisture and maceration of the skin.
- Drugs e.g. prolonged use of systemic corticosteroids, antibiotics, and immunosuppressives.
- Chronic debilitating diseases e.g. diabetes mellitus, lymphoma, and carcinoma.
- AIDS.

Clinical manifestations

Oral candidiasis (thrush). It is characterized by a sharply defined patch of creamy white pseudomembrane, when removed leaves an underlying erythematous base.

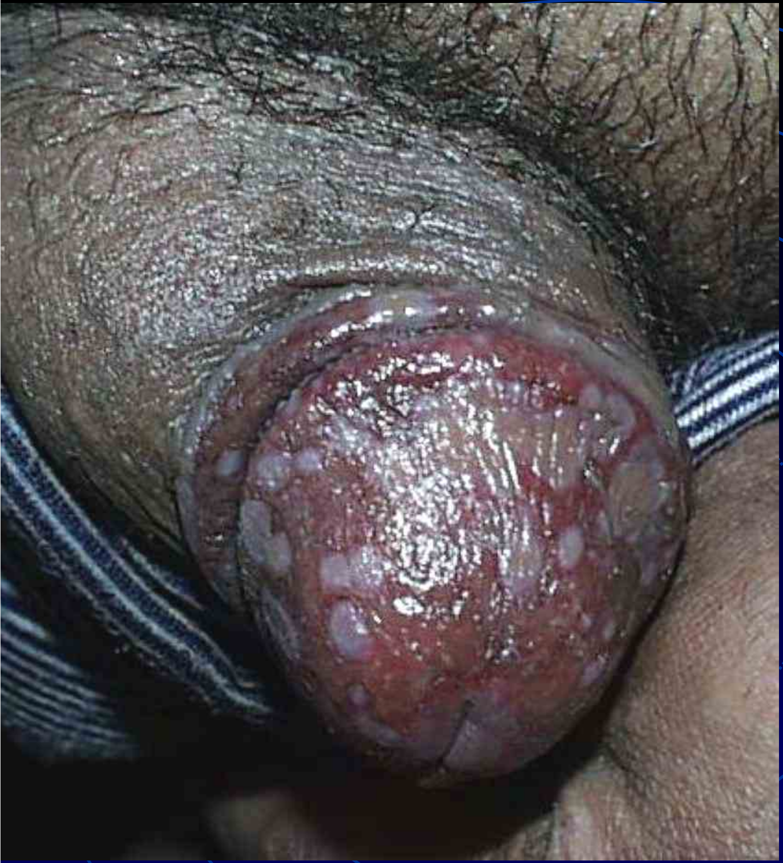
Perleche (angular cheilitis). It is a maceration fissuring of angles of mouth.

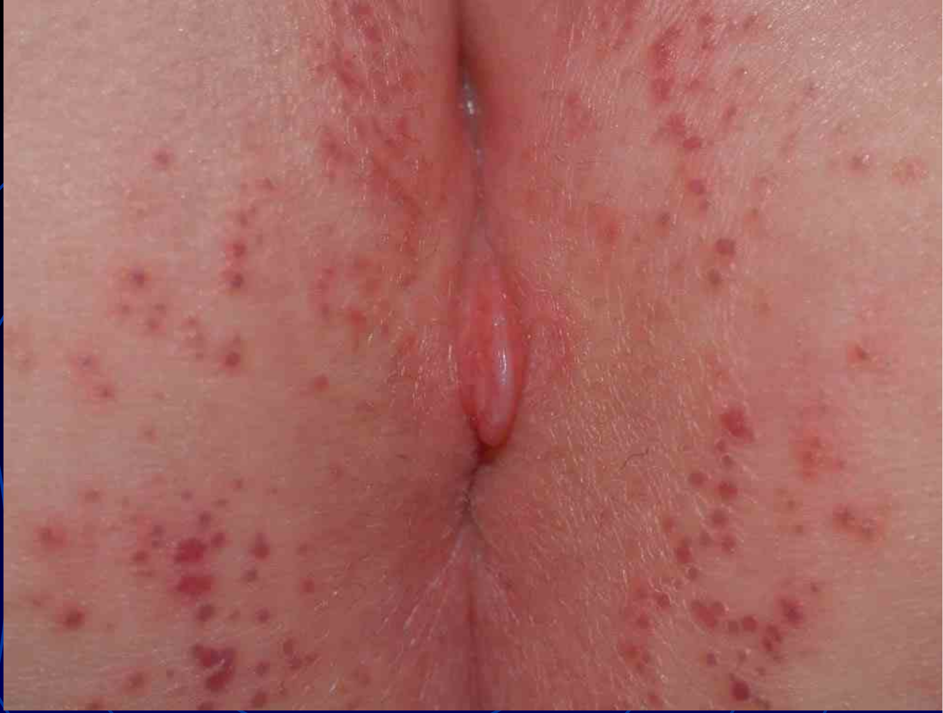
Candidal vulvovaginitis. The labia is erythematous, moist, and macerated and the cervix hyperemic, swollen, and eroded. The vaginal discharge is thick.

Candidal intertrigo. It affects skin folds, especially in the obese subjects.

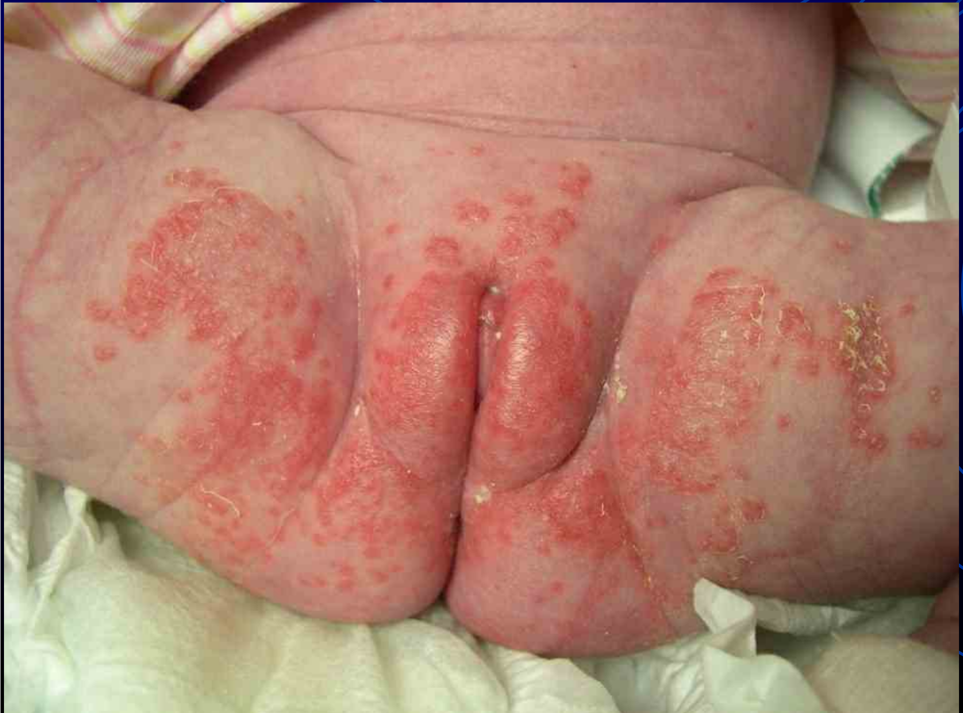
Napkin candidiasis. The dermatitis is usually enhanced by the maceration produced by the wet diapers.

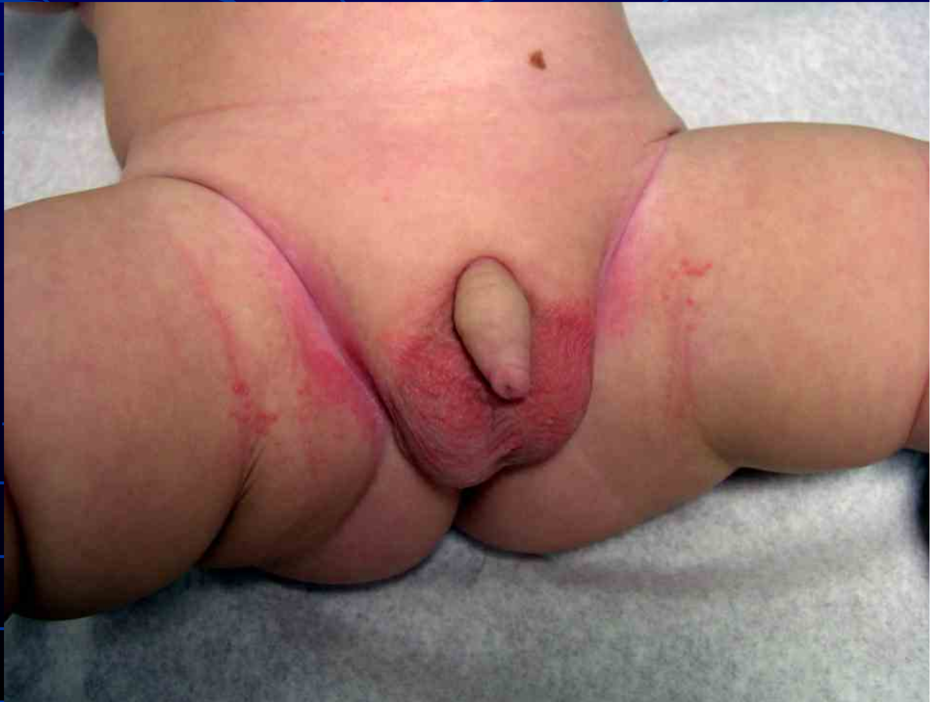
Candidal paronychia. It occurs as cushion-like thickening of the paronychial tissue, erosions of the lateral borders of the nail, gradual thickening and discoloration of the nail plates, and development of transverse ridges. Candidal paronychia occurs usually in those whose hands are frequently in water e.g. housewives and cooks.

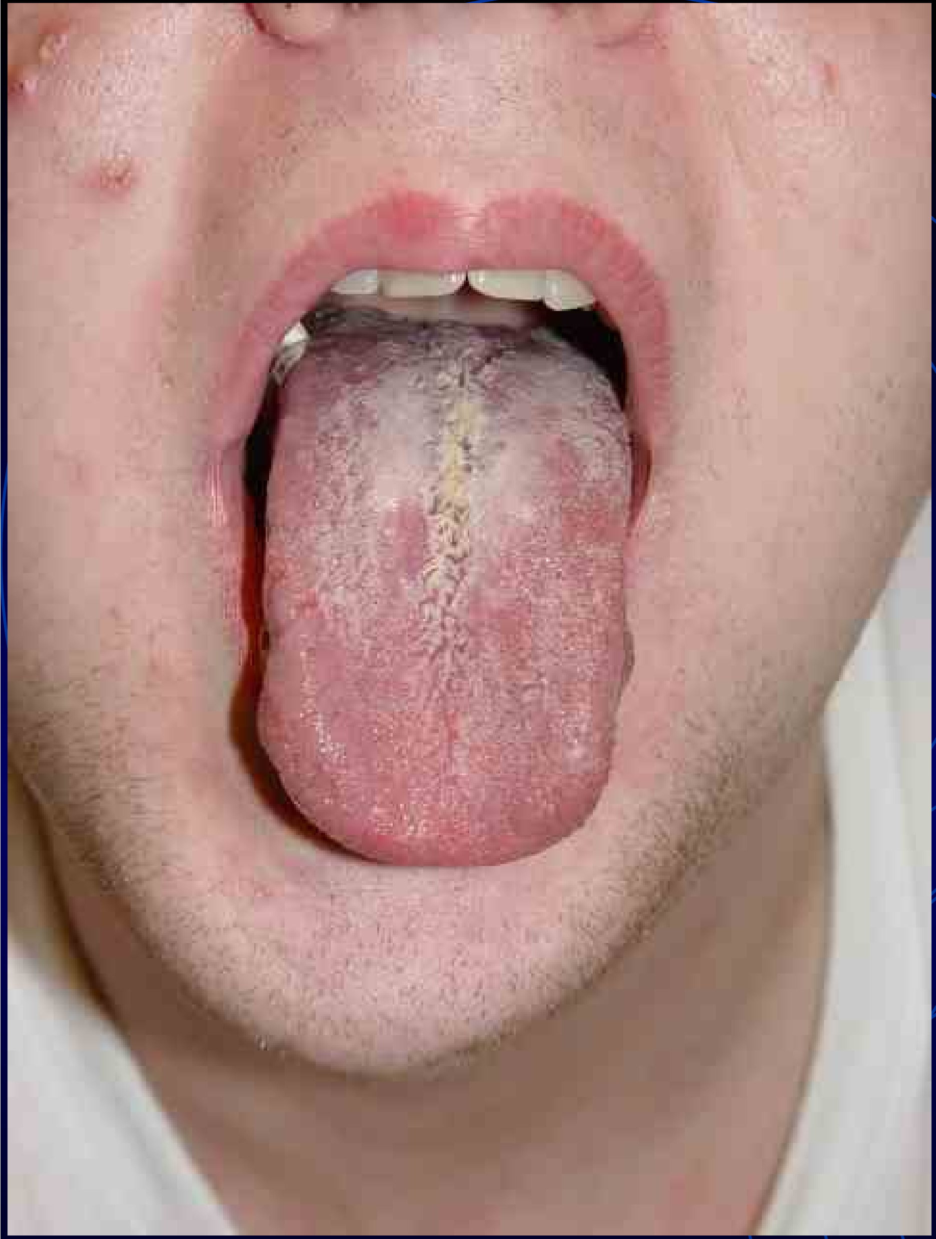


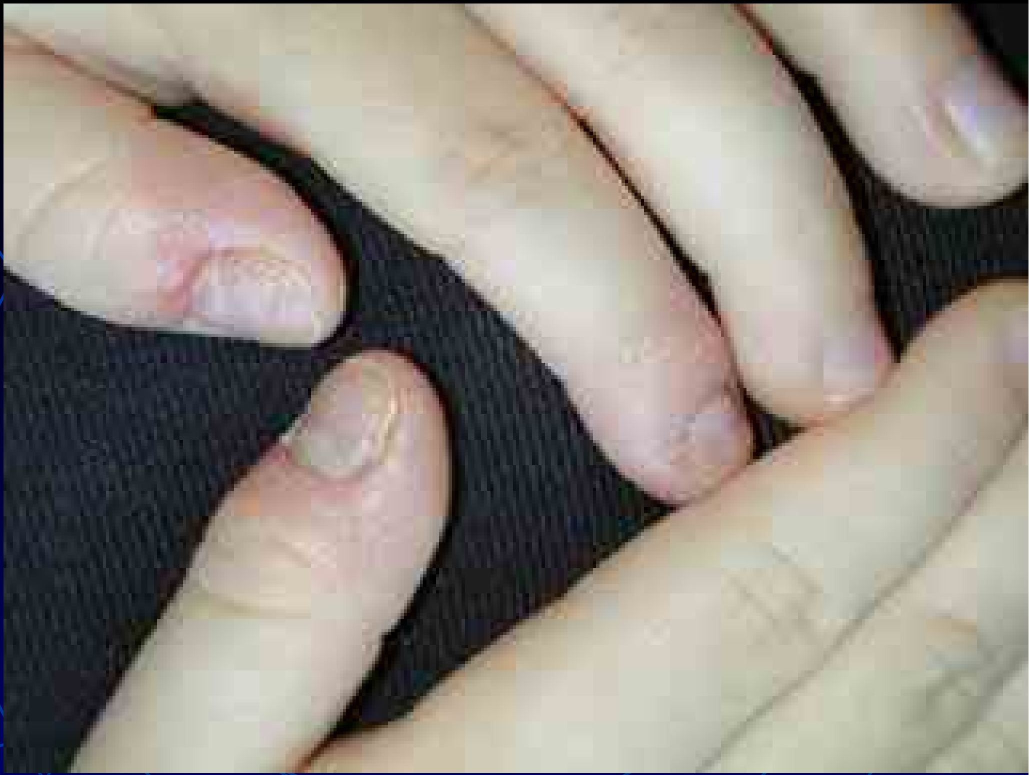


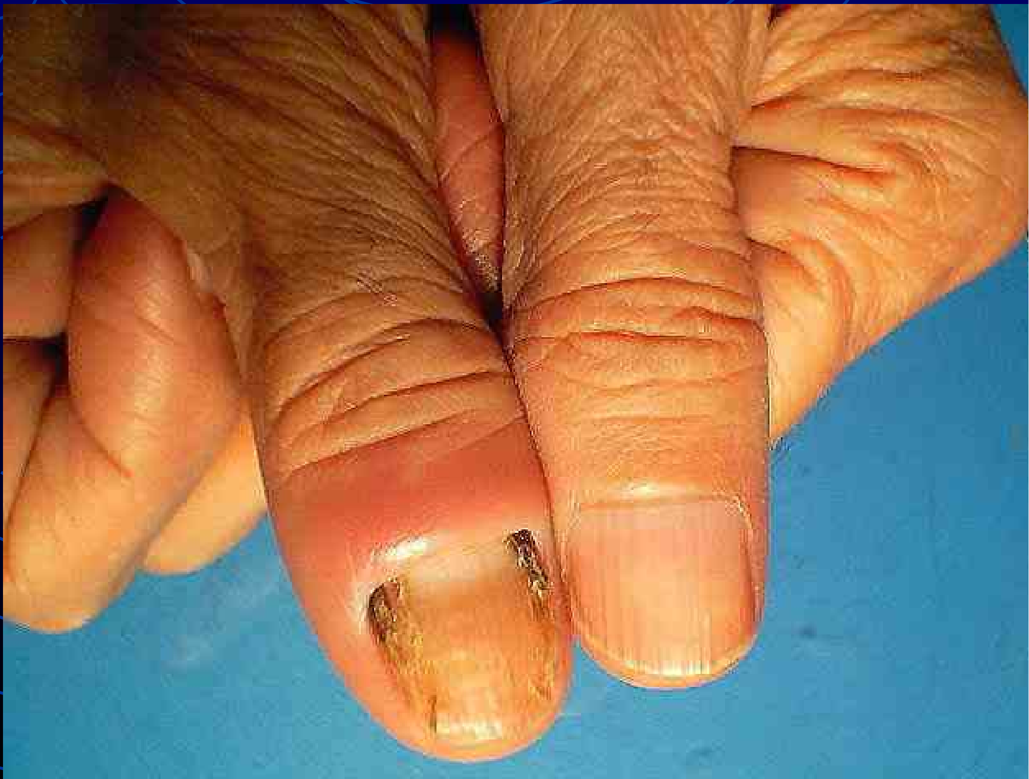
Candidiasis











Candidiasis

Treatment

- Topical nystatin.
- Topical imidazoles, as in the treatment of ringworm infections.
- Systemic treatment with ketoconazole, fluconazole or itraconazole.

Systemic therapies of fungi

Drug Oral	Disease and Dose		
	Ringworm	P. Versicolor	Candidiasis
Griseofulvin: 1. **Fulvin®** Oral (tablet 125 mg ❖ Syrup125mg/5ml). ❖ After fatty meal. ❖ Metabolized in liver ❖ Fungistatic. ❖ Only for ringworm. ❖	12.5 mg/kg/day: T. Capitis: 6-8 weeks. T. Corporis & cruris: for 2-6 week. T. Unguium: Fingers: 4- 6months. Toes: 6-12 months.	Not effective	Not Effective
Ketoconazole: 2. **Nizoral®** Oral (tablets 200 mg). ❖ Met. In liver. ❖ Liver toxicity. ❖ Fungistatic. ❖ Broad spectrum. ❖	One tab/day: T. capitis, corporis, cruris & pedis: for 2- 8 weeks. T. unguium: Fingers: 4- 6 months. Toes: 6 – 18 months.	1 – 2 tab/day: for 5-10 days. Then 1 tab/day / 3 days each month for 3 - 6 Ms.	Vulvo-vaginitis: 2 tab/day for 5 days.

Drug Oral	Disease and Dose		
	Ringworm	P. versicolor	Candidiasis
Itraconazole: **Sporanox®** Oral capsules 100 mg. Met. In liver. Fungistatic. Broad spectrum.	T. capitis, cruris, pedis: 1 cap/ day for 15-30 days. T. unguium: 2cap/twice/daily (7 days each months for 3 Month)	2 cap. Daily for 5-10 days.	Vulvo-vaginitis.: 2 caps/twice/ for one day only.
4. **Fluconazole** **Diflucan®** Oral: cap 50 &150 mg. Syrup: 25mg/5ml. Fungistatic . Broad spectrum.	T. capitis, cruris, pedis: 1cap weekly for 3-4 w. T. unguium: 1 cap/weekly for 6-9 months.	2 cap. Oral single dose. Repeated after 2 weeks.	Vulvo-vaginitis.: One cap oral single dose.
5. **Terbinafine:** **Lamisil®** Oral tab 125 & 250 mg. Met. In liver. Fungicidal. Broad spectrum.	T. cap, corporis, cruris & pedis: 1 tab (250mg) daily for 3-4 weeks. T. unguium: 1tab/day (250mg): Fingers: for 9 weeks. Toes: for 12 weeks.	**Not effective**	**Not effective against candida albica**

THANK YOU

Mycobacterial Infections

Abdelwasa Almekhlafi,MD

Tuberculosis of the skin

Classification

Inoculation tuberculosis (exogenous source)

- Tuberculous chancre (primary complex).
- Warty tuberculosis (tuberculosis verrucosa cutis).
- Lupus vulgaris (some).

Secondary tuberculosis (endogenous source)

- Scrofuloderma.
- Orificial tuberculosis.

Hematogenous tuberculosis

- Acute miliary tuberculosis.
- Lupus vulgaris (some).
- Tuberculous gumma.

Lupus vulgaris

Lupus vulgaris is the most common type of cutaneous tuberculosis and occurs in persons with a moderate or high degree of immunity.

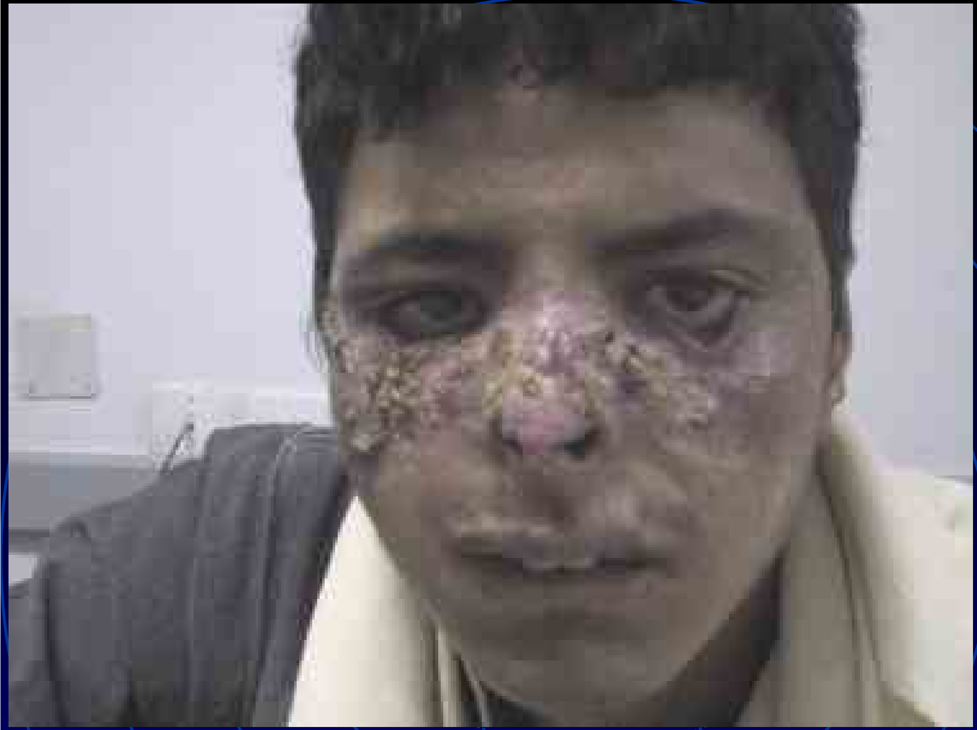
Clinical picture

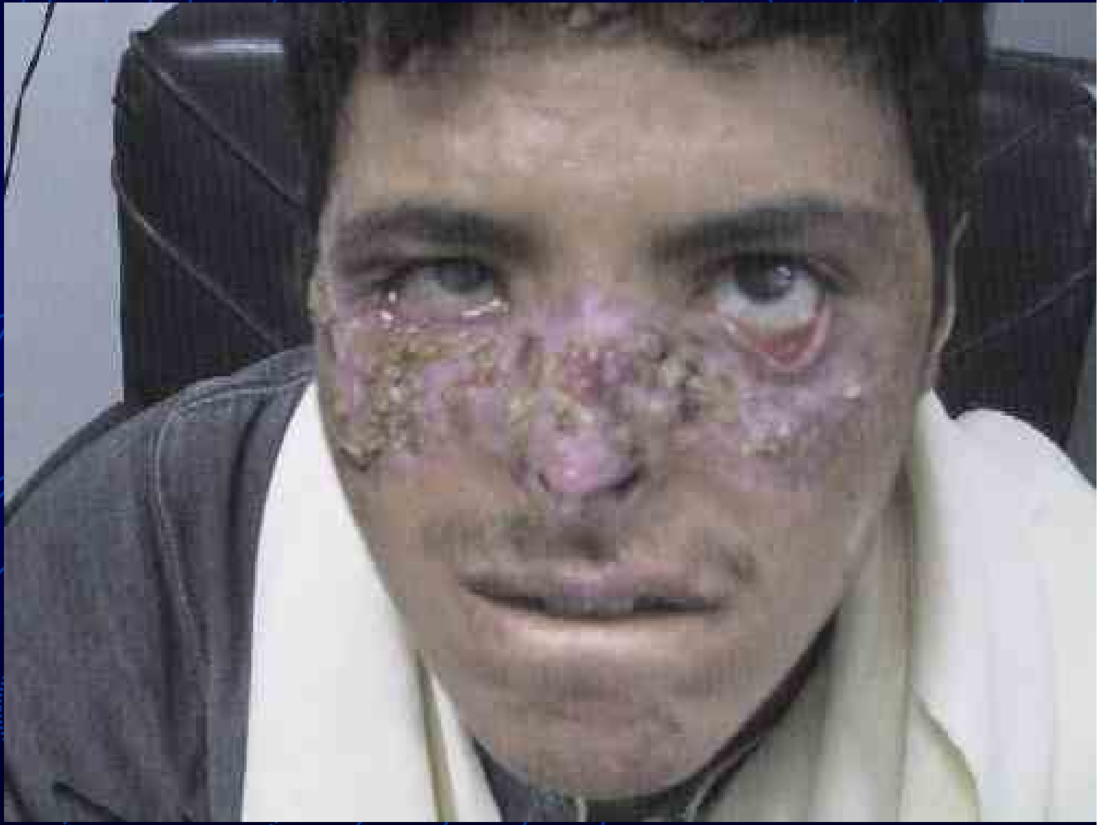
Lupus vulgaris is most prevalent on the face, especially the cheeks, upper lip and nose. The characteristic lesion is a plaque composed of soft reddish brown nodules (apple jelly nodules) with tendency to heal in one area and progress in another. The disease is destructive, frequently causes ulceration, and on involution leaves deforming scars as it slowly spreads peripherally over the years. Active lesions frequently appear in scar tissue (unhealthy scar).

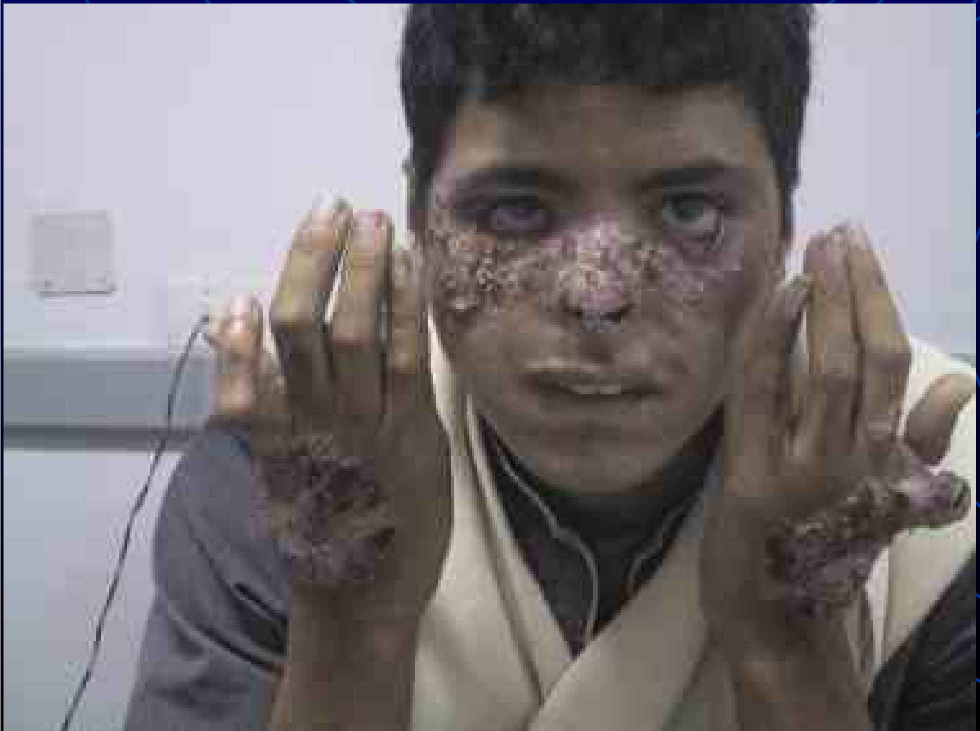
Complications

- Destruction of the underlying structures as nose or ear cartilage.
- Contraction may lead to ectropion or microstomia.

Lupus vulgaris





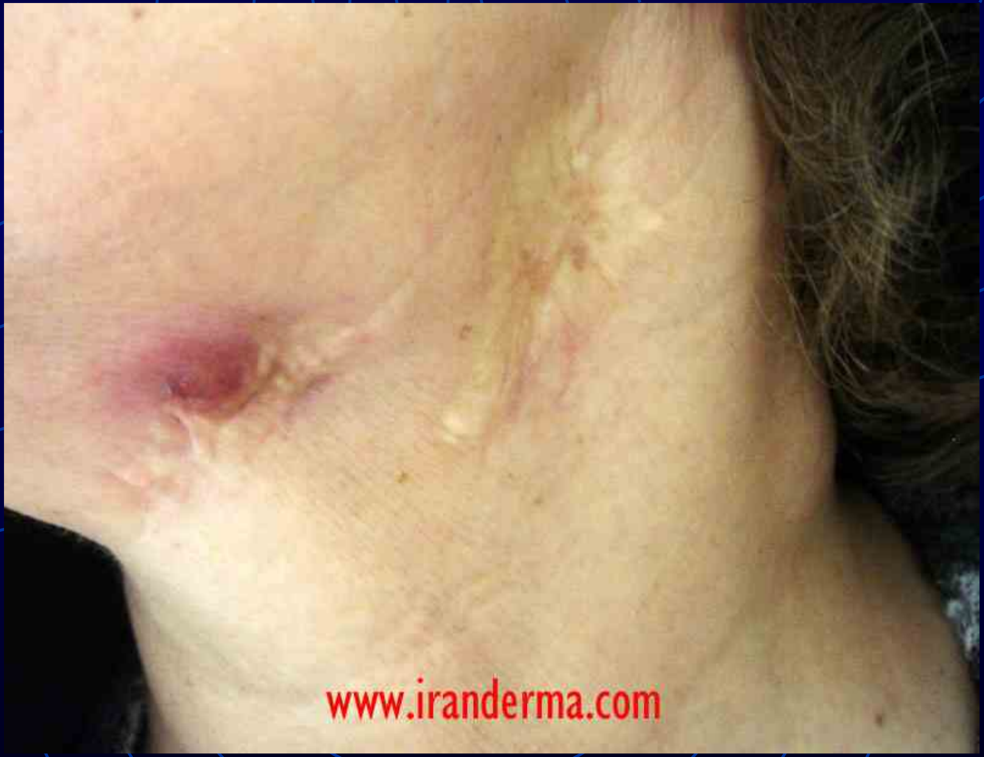


Scrofuloderma

- Scrofuloderma results from the involvement and breakdown of the skin overlying a tuberculous focus usually a lymph gland but sometimes an infected bone or joint.

- The process usually begins with a deep purplish induration of the skin overlying diseased lymphatic glands, which have been matted together. The glands break down and the resultant purulent and caseous exudates stretch the superimposed skin and form fistulae in it.

- Chronic discharging sinuses, ulcerations, granulations, crusts, hypertrophic scars and


www.iranderma.com

Treatment

A standard 6-month regimen for adults is now recommended. It includes the following 4 drugs:

- Isoniazid, 300 mg daily for the full 6 months.
- Rifampicin, 600 mg daily for the full 6 months.
- Pyrazinamide, 2-2.5 g daily for the first 2 months.
- Ethambutol, 15 mg/kg daily for the first 2

Leprosy

Leprosy (Hansen's disease) is a chronic, systemic infectious disease, affecting primarily the peripheral nerves and secondarily the skin, mucous membranes, the eye, bones and viscera.

Etiology

The causative agent of leprosy is the *Mycobacterium leprae.* It is usually contracted during childhood as adults are relatively non–susceptible. Both sexes are generally affected, lepromatous leprosy is more common among men.

Incubation period 2-5 years for tuberculoid and 8-12 years for lepromatous cases.

Mode of infection Prolonged close contact of susceptible individuals to an open case of leprosy (untreated patients with multibacillary type with positive nasal smears) is very important. Nasal discharge from the highly infectious

Classification

Based on the clinical, bacteriologic, immunologic and histopathologic features, leprosy is classified into 5 types:

- Tuberculoid leprosy (TT).
- Borderline tuberculoid leprosy (BT).
- Mid-borderline leprosy (BB).
- Borderline lepromatous leprosy (BL).
- Lepromatous leprosy (LL).
- A sixth type not included in the classification is the ‘**indeterminate**’ leprosy.

According to the results of slit - skin smears, leprosy is classified into two types:

- *Paucibacillary,* with scanty or absent bacilli (TT, BT).
- *Multibacillary,* with numerous bacilli (BB, BL, LL).

Diagnosis

- Clinical .
- Slit-skin smears.
- Skin biopsy.
- Nerve biopsy.
- Lepromin test.

Tuberculoid leprosy

This type affects only nerves and skin, and may be purely neural (*neural leprosy*).

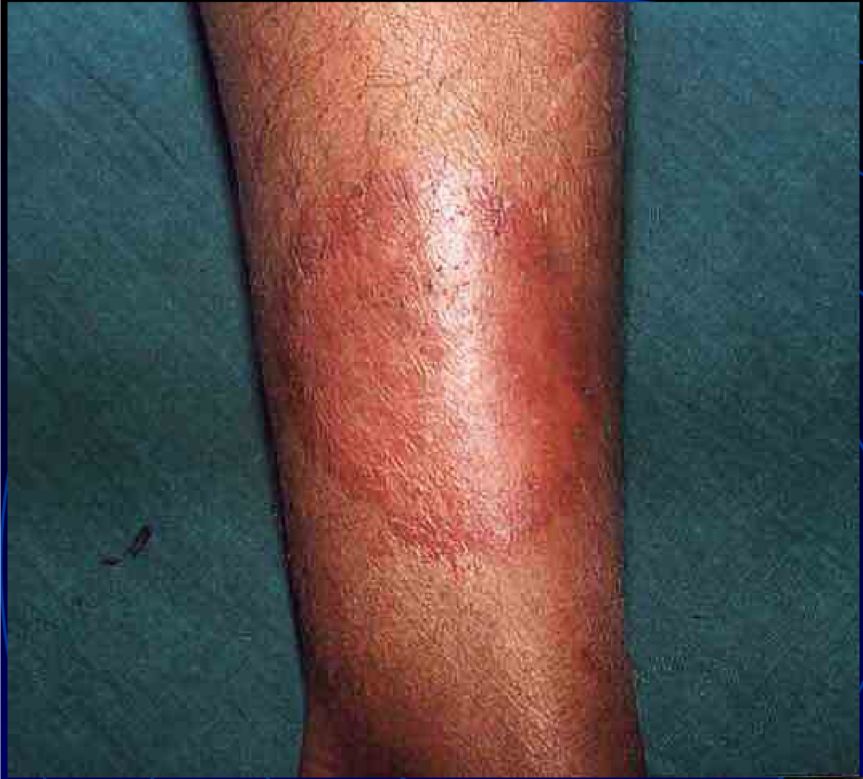
Cutaneous lesions are few, often solitary, with asymmetrical distribution and occur on the face, limbs or anywhere. The typical lesion is an erythematous plaque with raised and clear-cut edge sloping towards a flattened and hypopigmented centre. The surface of the lesion is usually dry (anhidrotic), hairless and anesthetic.

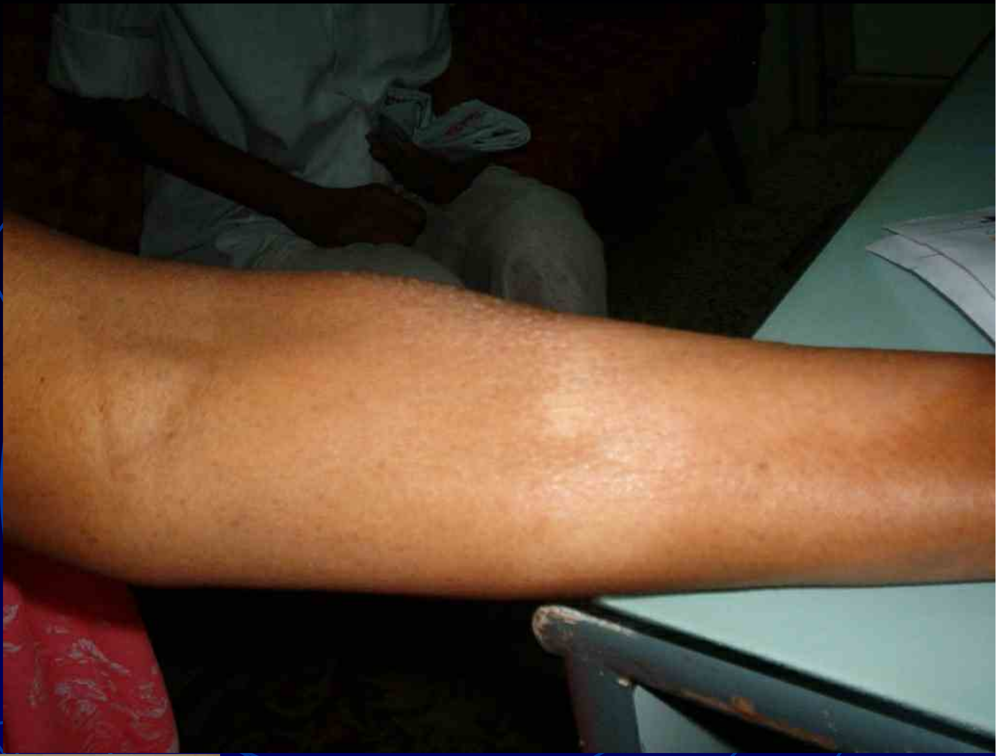
Nerve involvement is usually marked and in a few nerves.

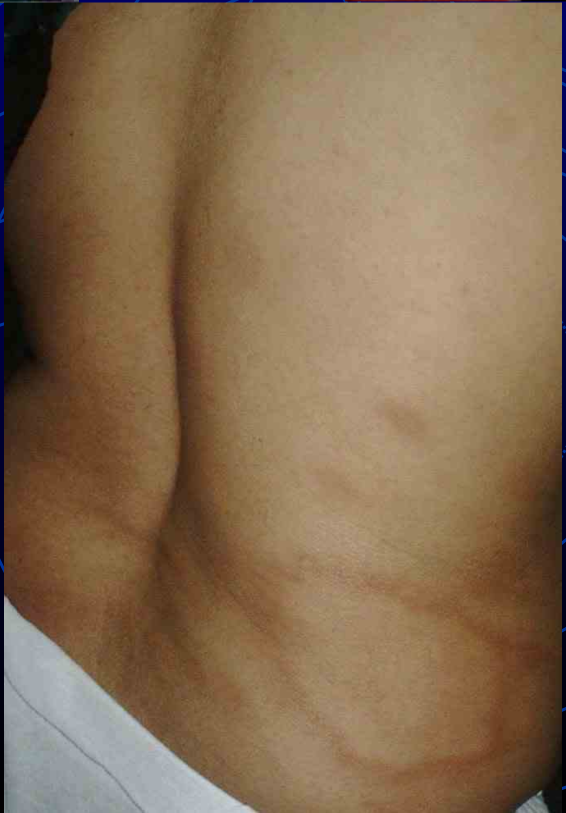
The bacilli are usually absent in slit-skin smears.

The histopathology shows tuberculoid granulomas composed of epithelioid cells surrounded by a zone of lymphocytes.

Lepromin test is strongly positive







leprosy

Lepromatous leprosy

- In this type, skin, nerves, mucous membranes, eyes, bones and internal organs are involved.

- Cutaneous lesions consist of macules, papules, infiltration or nodules (lepromas). They are numerous, bilateral, symmetrical, ill-defined with shiny surface. The sites commonly affected are the face, arms, legs and buttocks, but may be anywhere. Hair growth, sweating and sensation are not initially impaired over the lesions. Infiltration and deepening of the lines of the forehead lead to the characteristic *"leonine facies"* . The ear lobes may be infiltrated and thickened.

- A slow progressive loss of hair takes place from the outer third of the eyebrows, then the eyelashes.

- Mucous membranes are commonly involved in the form of papules, nodules and ulcerations. The nasal mucosa is hyperemic or ulcerated and bleeds easily; epistaxis is

- Eye involvement is also common in the form of keratitis, iridocyclitis, pannus formation and corneal lepromas.

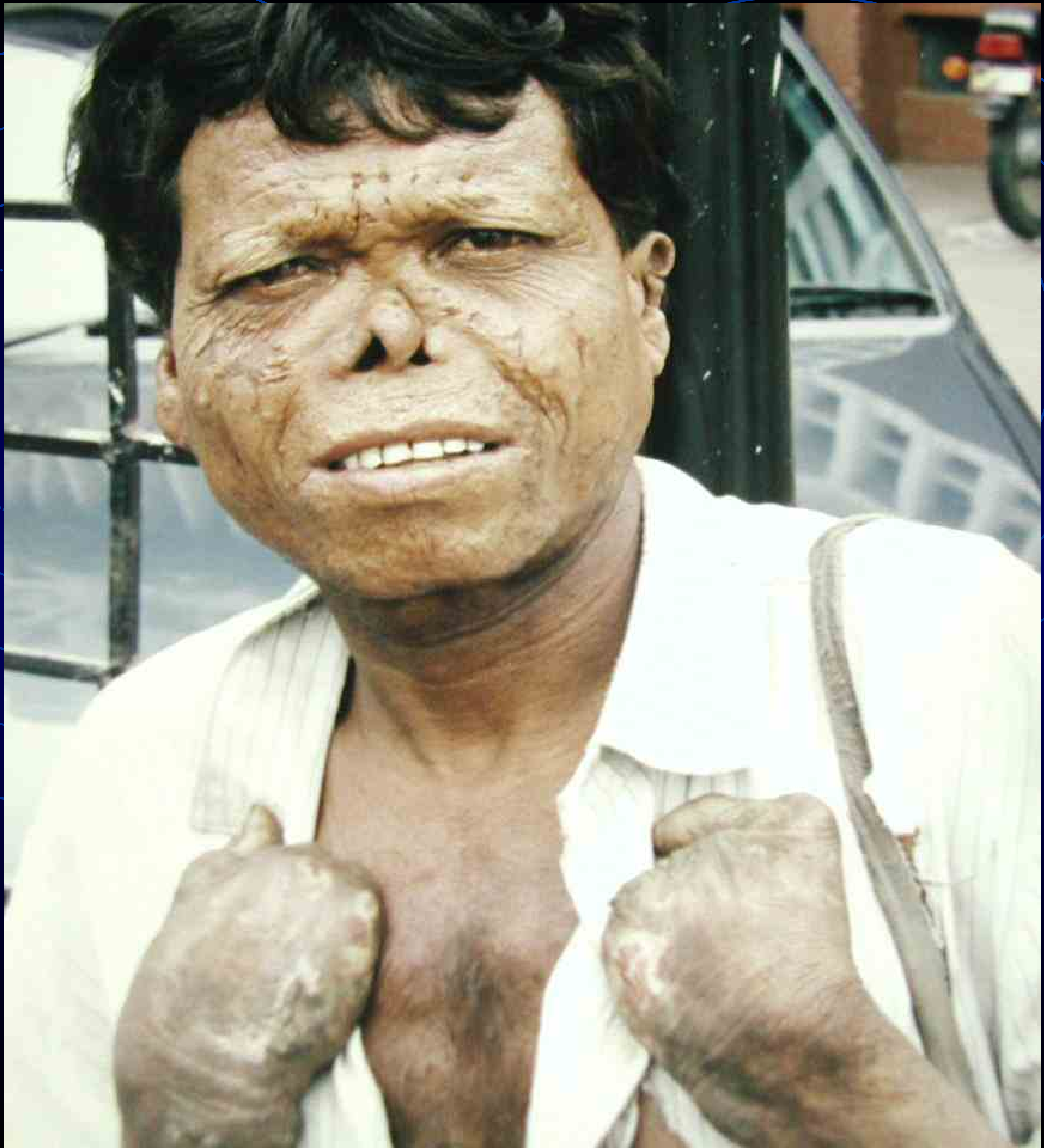
- Testicular atrophy causes infertility, impotence and gynecomastia.

- Nerve involvement is widespread but slight as it develops very slowly, and usually bilateral and symmetrical, unlike those of TT leprosy.

- The bacilli are usually abundant in slit- skin smears.

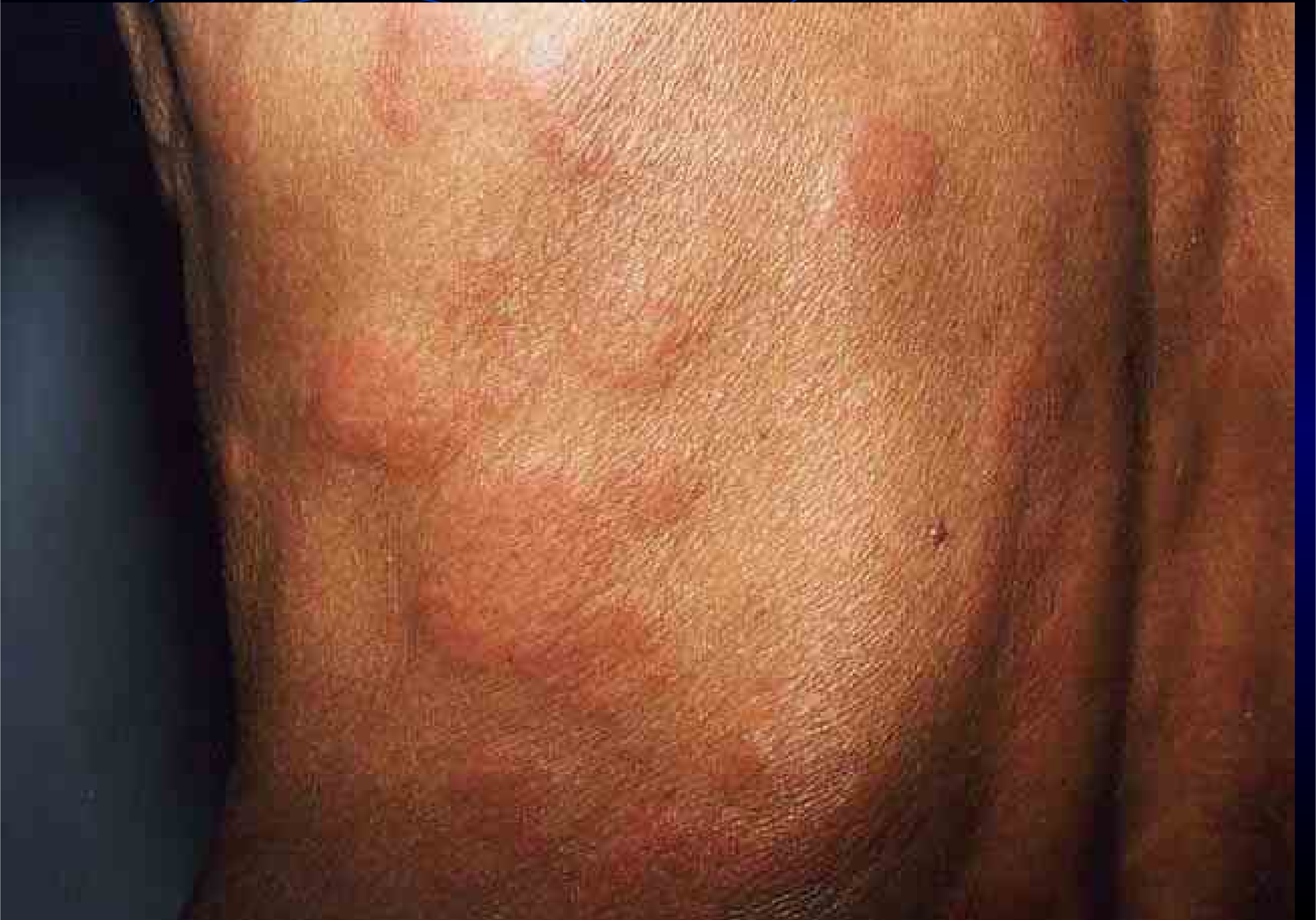
- Histopathology shows granulomatous lesions composed chiefly of bacillus-laden histiocytes (foam or lepra cells).

- Lepromin test is negative.



Borderline leprosy

- Skin lesions are intermediate in number between those of the two previous polar types. They may take the form of macules, plaques and annular lesions. Plaques with a "punched-out appearance" are characteristic of the mid-borderline type.
- Nerve involvement is early, affects several nerves and asymmetrical.
- Bacilli are present in slit-skin smears but less than in lepromatous leprosy.
- Histopathology shows mixed tuberculoid & lepromatous features.



Reactions in Leprosy

During the chronic course of leprosy, acute episodes (reactions) may occur. There are 2 main types of reactions, type 1 and type 2.

Type 1 reaction

- It occurs in borderline leprosy.
- It is due to the rapid change in cell - mediated immunity.
- Nerves become swollen and tender with loss of sensory and motor functions. Serious complications as facial palsy and dropped foot may occur.
- Existing skin lesions become erythematous, edematous and may ulcerate.

Type 2 reaction (erythema nodosum leprosum)

- It occurs in patients with LL and BL.
- It is an immune - complex disorder.
- ENL manifests as painful, red nodules on the face and extensor surfaces of limbs, and fading over several days.
- Fever and malaise are common and may be accompanied by uveitis, arthritis, lymphadenitis, myositis and orchitis.

Treatment

The first- line antileprosy drugs are rifampicin (bactericidal) dapsone (weak bactericidal) and clofazimine (weak bactericidal and anti–inflammatory). The following is the recommended WHO multidrug therapy regimen.

1- Paucibacillary leprosy

- Rifampicin, 600 mg monthly supervised.
- Dapsone, 100 mg daily self-administered.
- Duration of treatment: at least 6 months.

2- Multibacillary leprosy

- Rifampicin, 600 mg monthly supervised.
- Dapsone, 100 mg daily self-administered.
- Clofazimine (Lamprene), 300 mg monthly supervised + 50 mg daily self-administered.
- Duration of treatment: at least 2 years.

Treatment of reactions

- The antileprosy drugs should be continued without interruption.
- Rest, analgesics and anti-inflammatory drugs.
- Clofazimine in large doses (300 mg / day).
- Corticosteroids, used in moderate and severe cases.
- Thalidomide has a dramatic effect in controlling ENL but is

PARASITIC SKIN INFECTION

SCABIES

PEDICULOSIS

PARASITIC
SKIN
DISEASES

LEISHMANIASIS

ONCHOCERCIASIS

Scabies

Scabies is a common contagious disease caused by infestation with a mite called *Sarcoptes scabiei.* The fertilized female parasite is responsible for the infestation. It invades the stratum corneum and forms a burrow where it deposits its eggs. These hatch to give larvae and continue the life cycle. The incubation period is 2 weeks.

Mode of infection

- Close contact with the patient.
- Indirect, by using clothes or bed of the patient.
- Contact with infected animals, as cats, dogs or rabbits

Clinical picture

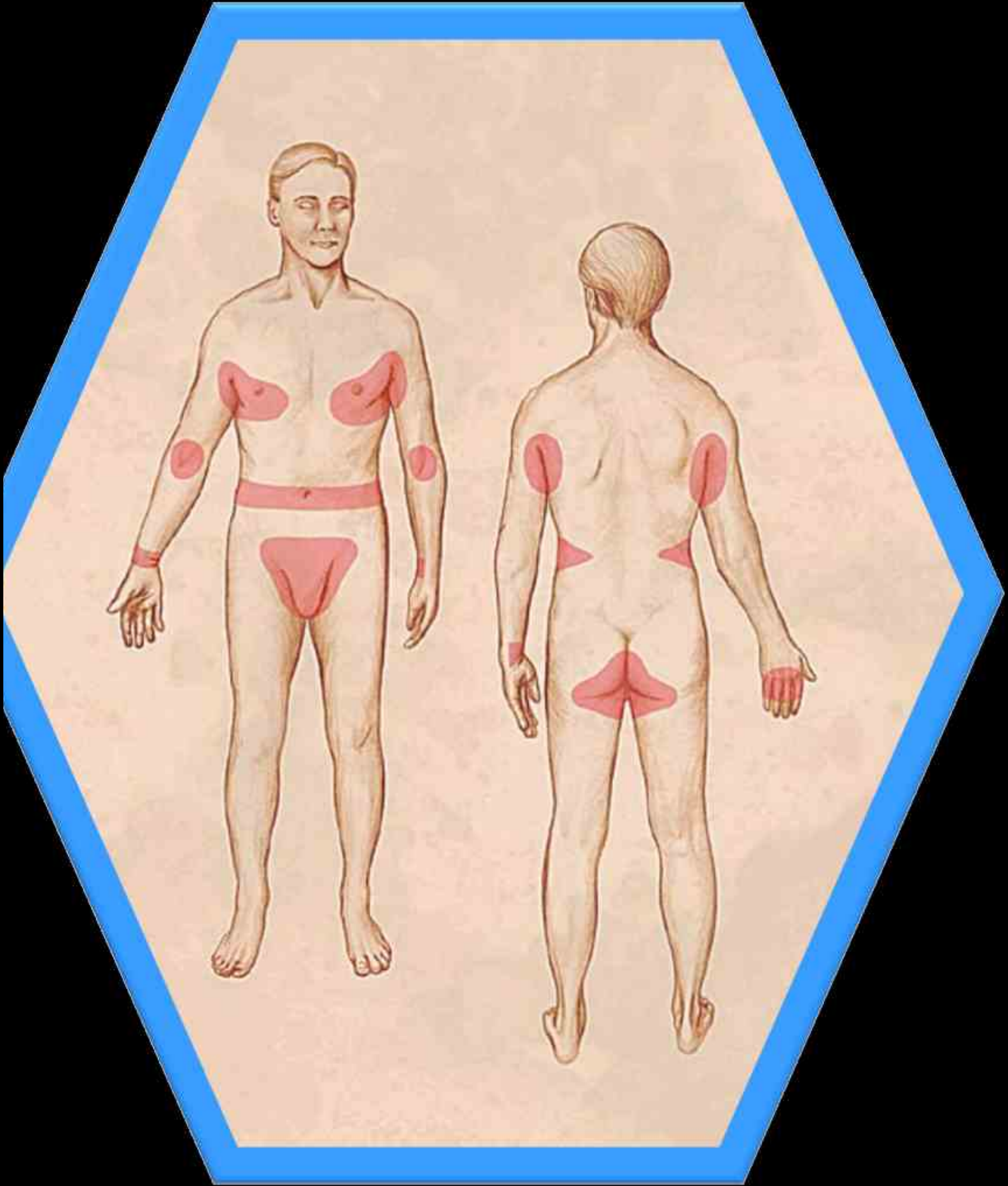
- Severe itching, especially at night.
- The lesions consist of burrows, which are the primary and characteristic lesions of scabies. Burrows are skin-colored or grey curved ridges where the mite resides. Other lesions include papules, vesicles, pustules and scratch marks.
- Sites of predilection are: interdigital webs, sides of fingers, wrists, medial side of the forearms, elbows, cubital fossae, axillary folds, under the breasts and around the nipples (in females), around the umbilicus, lower abdomen, genitalia, buttocks, popliteal fossae, medial aspect of thighs and legs and

Varieties

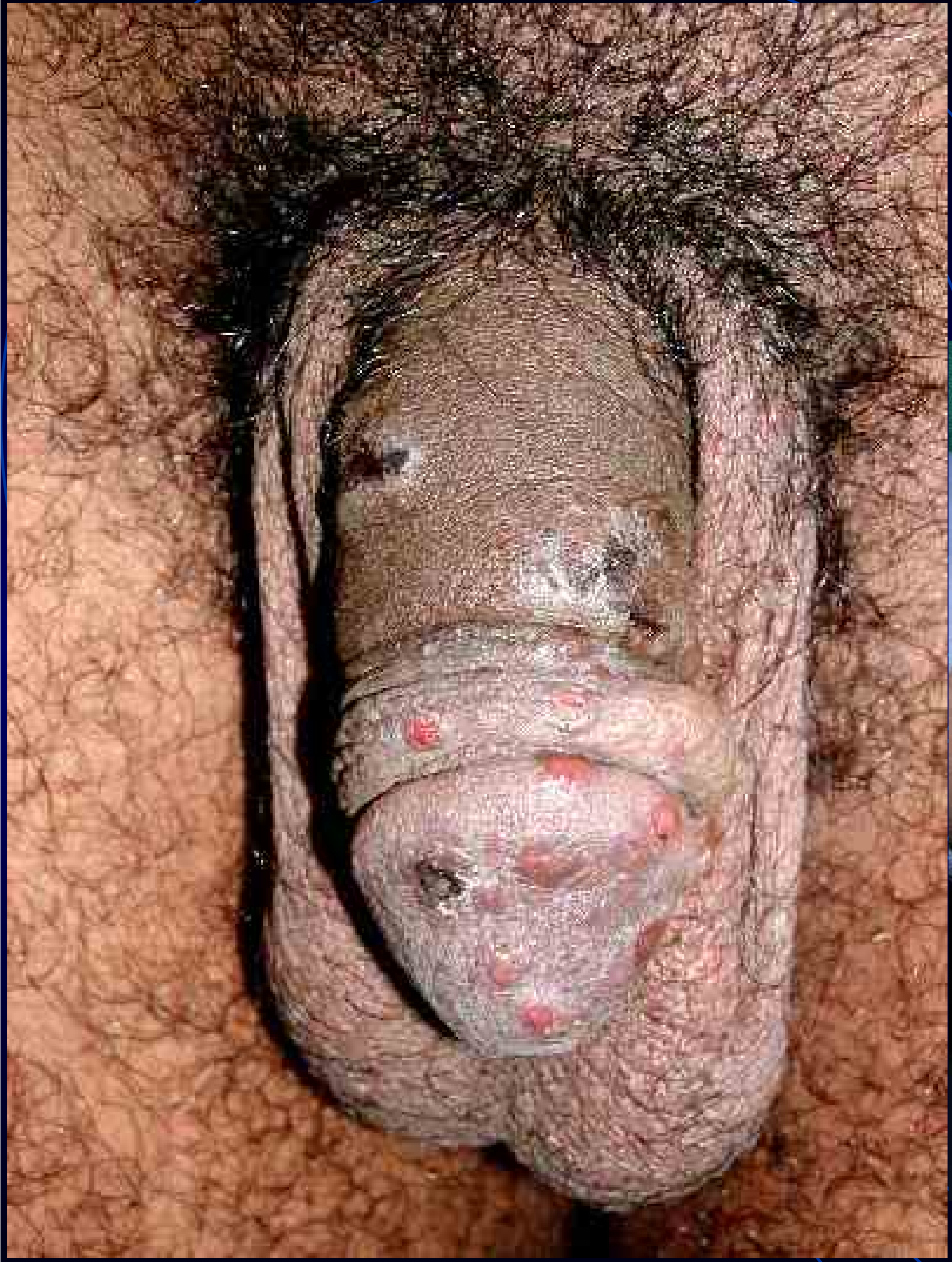
- **Scabies in infants.** It differs from adults in:
- Atypical distribution to include head, neck, palms and soles.
- Extensive distribution of burrows.
- Vesicles and vesiculopustules are frequent.
- Frequent secondary bacterial infection.
- Frequent eczematization.
- **Scabies in the clean.** Burrows are difficult to find due to frequent bathing.
- **Scabies incognito.** Inappropriate use of topical steroids may modify the clinical picture to simulate other skin disorders.
- **Nodular scabies.** Itchy indurated, reddish-brown nodules usually seen on scrotum or penis and persist for weeks or months after treatment, leading to persistent itching.
- **Animal scabies**. Differs from human scabies in:
- - Short incubation period - Absence of burrows
- - Not transmitted from human to human. - Self-limiting

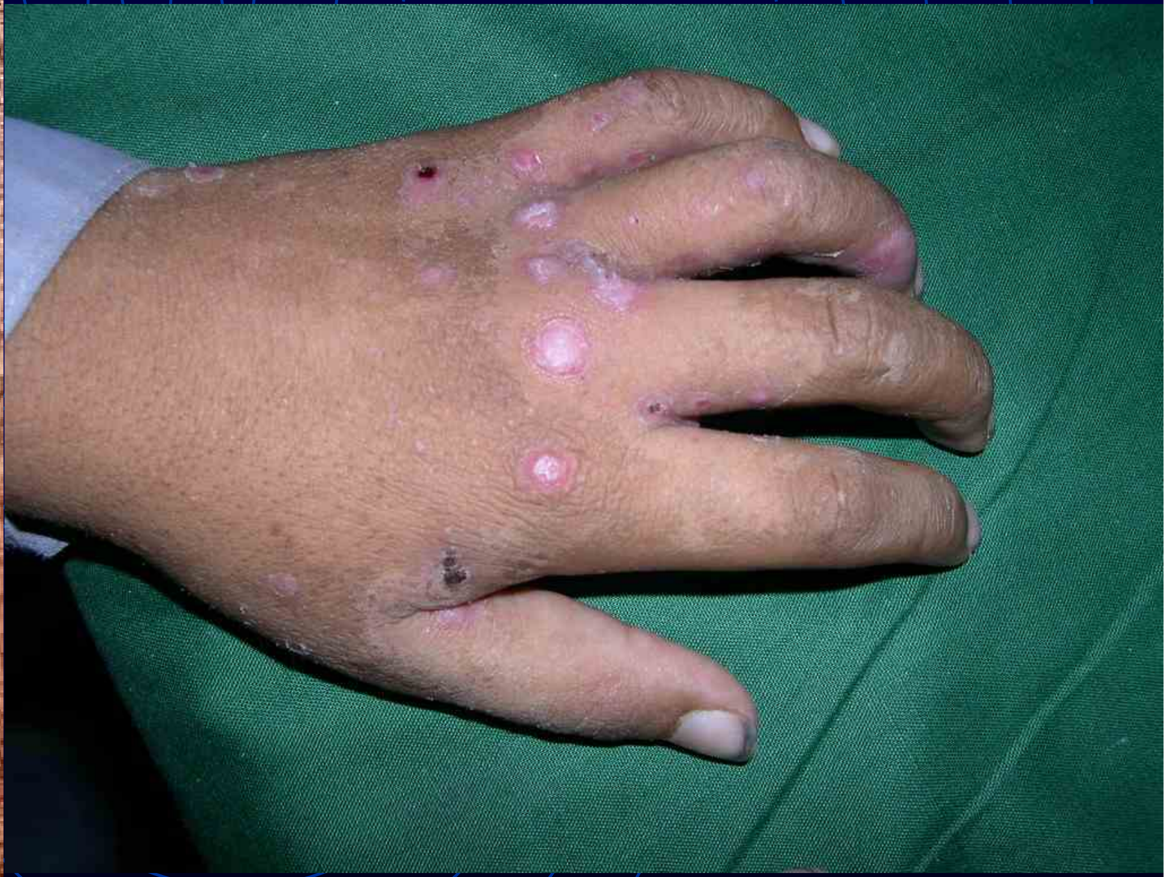
Scabies

Common sites



Scabies





Complications

- Secondary bacterial infection.
- Eczematous changes
- Acarophobia: occurs in neurotic patients where the symptoms continue after successful treatment due to fear from persistence of the infestation.

Diagnosis

- Itching mainly at night.
- Presence of burrows.
- Distribution of the lesions.
- Presence of contact cases within the family.
- Microscopic demonstration of the parasite.

Differential diagnosis

- Papular urticaria
- Prurigo
- Eczema

Treatment

Topical treatment

Topical scabicides should be applied to body, except the head after a hot bath:

- **Sulfur ointment 3-10%** It is applied for 3-4 successive nights. The ointment is safe and effective but rarely used because it is messy, staining and causes irritation and dermatitis.
- **Benzyl benzoate 25%.** It is used as one night treatment but better applied for 3-4 nights.
- **Gamma benzene hexachloride 1%.** It is used as one night treatment. It should be avoided for infants, children and pregnant women because of neurotoxicity.
- **Permethrin 2.5-5%.** It is the safest and most effective and applied for one night.
- **Crotamiton (10%).** It is applied for 3 successive nights followed by a bath in the 4th day. Crotamiton is less effective than the previous scabicides but has an antipruritic effect.

Systemic treatment

- Antihistamines for itching. Antibiotics for secondary infection.
- Intralesional injection of steroids for scabitic nodules.
- Ivermectin (Mectizan) a sigle oral dose of 200 mic g/kg

Pediculosis capitis

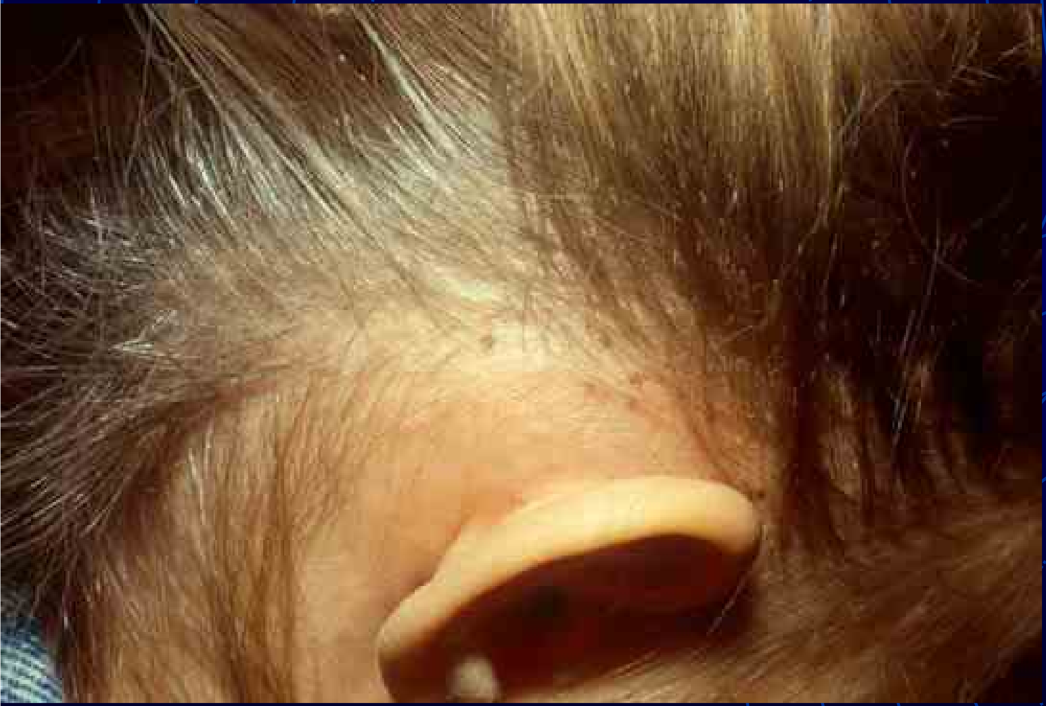
Pediculosis capitis is a common infestation which is usually transmitted by sharing head-covers, combs or brushes. It is common among school children, especially girls. The main symptom is pruritus. Secondary bacterial infection is common as a result of scratching.

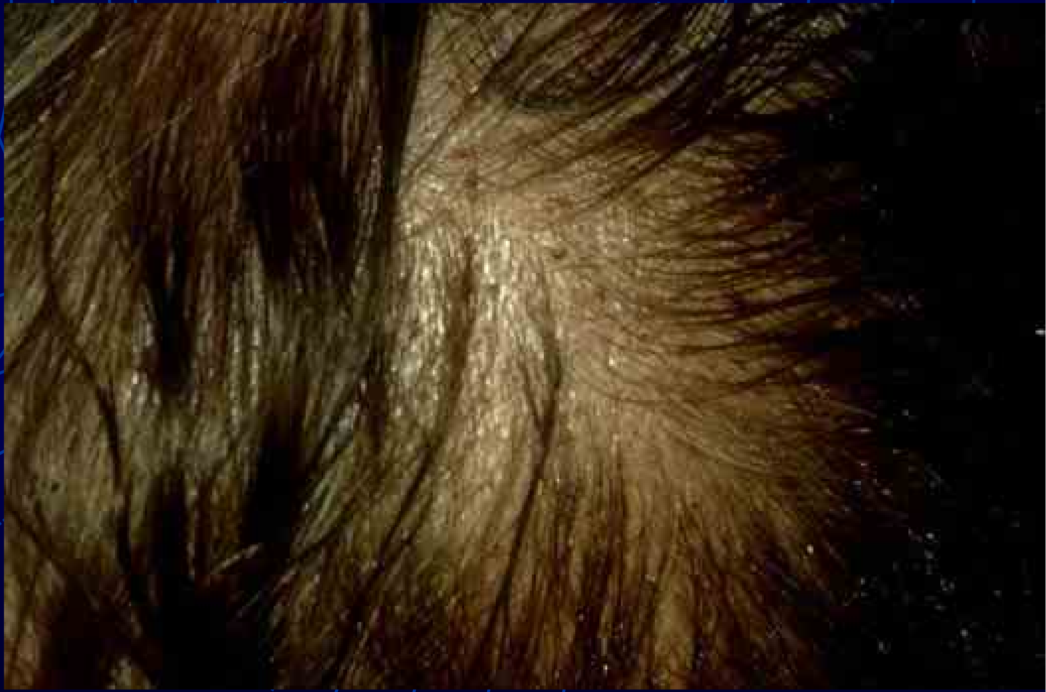
Diagnosis By finding the parasite or their eggs (nits).

Treatment

- **Permethrin (1%)** is the drug of choice. It is applied for 10 minutes and washed off.
- **Malathion (0.5%)** applied for 12 hours.
- **Gamma benzene hexachloride (1%)** shampoo to be rinsed out after 10 minutes.
- **Crotamiton (10%)** to be applied for 24 hours.
- Remaining nits may be removed by a fine-toothed comb or forceps.
- **Antibiotics** for secondary infection

Pediculosis capitis





CUTANEOUS LEISHMANIASIS

Leishmaniasis

Etiology: *Protozoa : Genus: Leishmania.*

Species:

1. *Leishmania Donovani: Causes:*
   - *Visceral leishmaniasis (Kala azar).*

2. *Leishmania Tropica: Causes:*
   - *Cutaneous leishmaniasis (Oriental sore).*

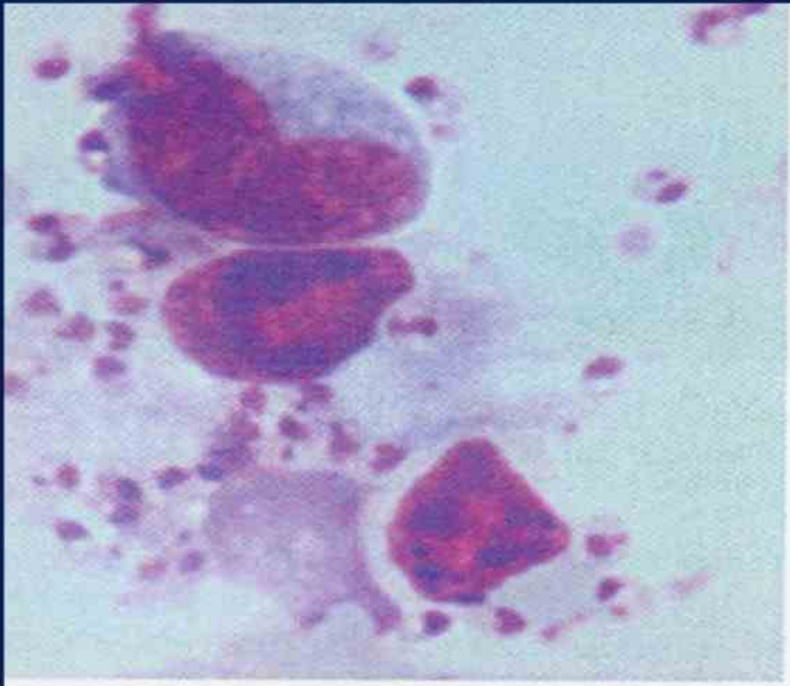
3. *Leishmania Braziliense: Causes:*
   - *Mucocutaneous leishmaniasis (Espundia).*

Leishmaniasis

All are morphologically identical. They have two forms:

- *Leishmania form (Amastigote): in man: oval or spherical, 2 - 3 μ, no free flagellum,* replicates in macrophage phagosomes in mammalian hosts, *but never found free in peripheral blood.*
- *Leptomonas form (Promastigote): 7 - 15 μ, spindle, has flagellum,* found in sandflies and culture. *in the gut of sandfly, culture media and extracellular.*

Leishmania Donovani
Amastigotes



-58 *Leishmania donovani* amastigotes.

Cutaneous leishmaniasis

Etiology:

1. *Leishmania major → Wet (Rural) acute leishmaniasis.*
2. *Leishmania tropica → Dry (Urban) sub-acute leish..*

Mode of infection:

- ☐ *By the bite of sandfly insect (the vector).*
- ☐ *By direct contact with infected material from the lesion.*
  - *Female sandfly (Phlebotomus): which:*
    - *Lives in low, dark places.*
    - *Active at night.*
    - *Soundless flying.*

Source of infection:

1. *Animals : as rodents and dogs.*
2. *Infected humans.*

Endemic areas:

Yemen (many areas), middle east, Mediterranean area, India, Africa.

Cutaneous Leishmaniasis
Clinical picture

- *Cutaneous leishmaniasis can effects any age and both sex.*
- *Site: On exposed parts: face, nose, ears, lips, hands, legs and feet.*
- *Lesion: indurated nodule (s)→ ulcerates → ulcer with undermined edge surrounded by a red zone, 2ry infection may occur, finally the lesion →heal with big disfiguring scar.*
- *The disease produce solid immunity.*
- *Clinical types:*

A. *Acute cutaneous leishmaniasis (ACL).*

B. *Chronic cutaneous leishmaniasis (CCL).*

C. *Leishmaniasis Recidivans (LR).*

D. *Diffuse cutaneous leishmaniasis (DCL).*

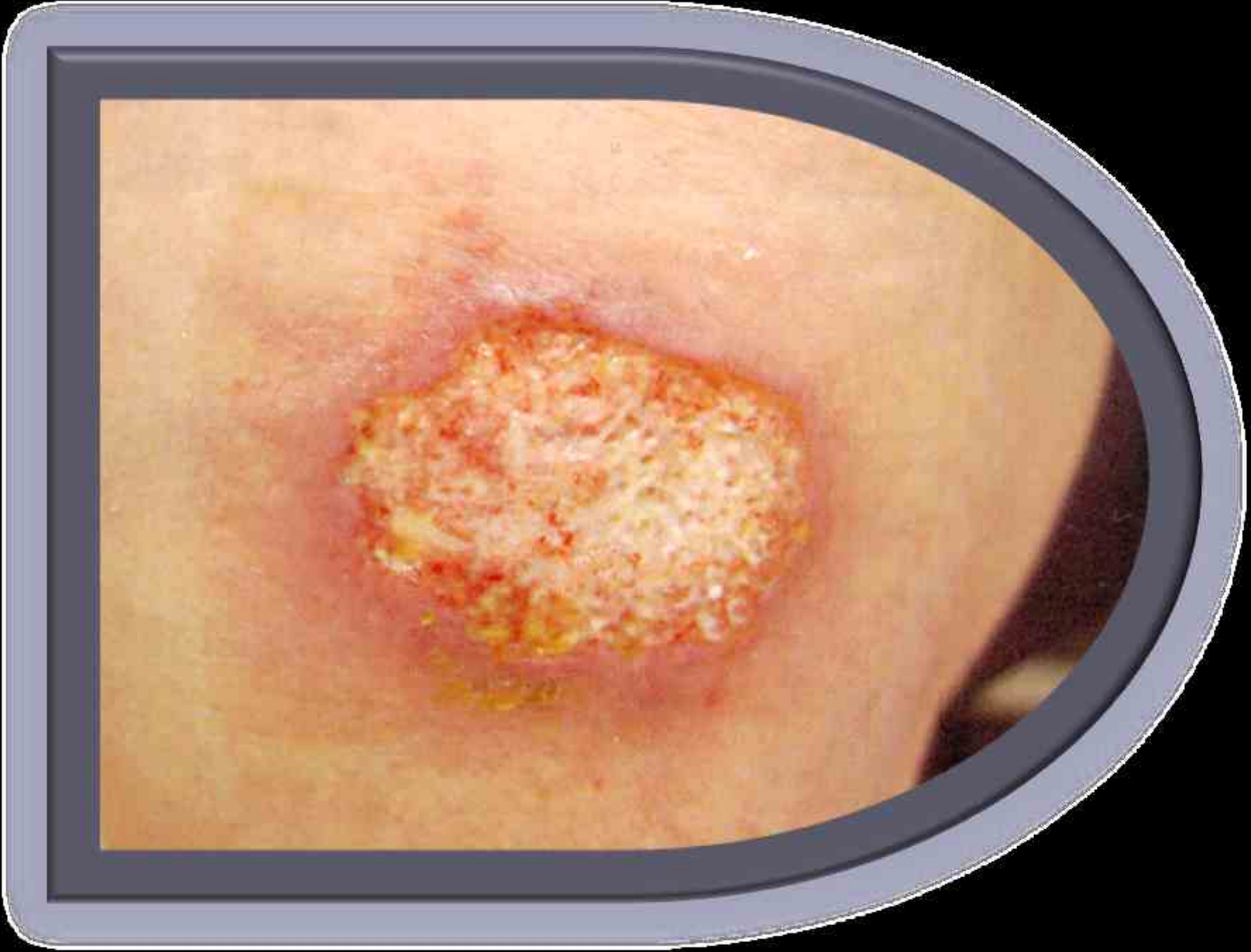
Cutaneous Leishmaniasis

A. Acute Cutaneous Leishmaniasis

	Wet (Rural) Early Ulcerative type	Dry (Urban) Late Ulcerative type
Etiology	L. Major	L. Tropic
Source	Rodents.	Humans, dogs.
Vector	Phlebotomus Papatassi.	Phl. Sergenti.
Incubation period	Less than two months.	More than two months.
Clinically Lesion (single or multiple)	Nodule (red)→ furuncle-like. • In 2 weeks → Crust • Crust fall → Ulcer • (large with red margin).	Nodule: small, brownish. • In 6 months→ plaque (1-2 cm). • → Shallow ulcer at the center→ covered by adherent crust.
Healing→ Scar	2 – 6 months.	8 – 12 months.

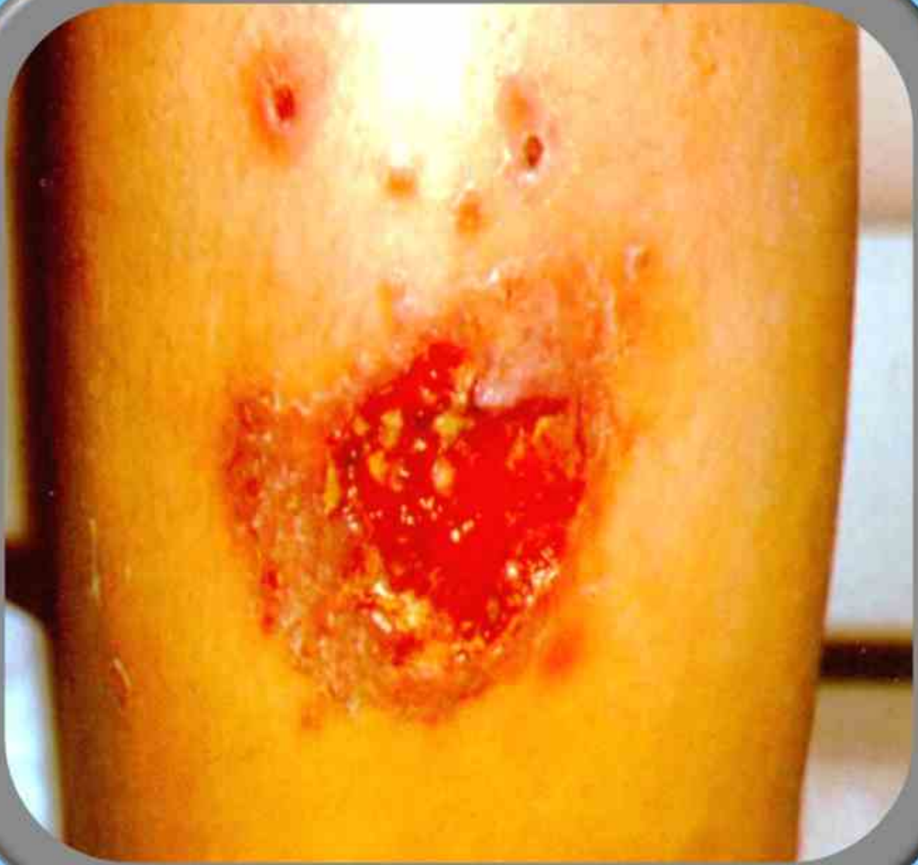
Cutaneous Leishmaniasis

Leg: Ulcer with undermined edge



Cutaneous Leishmaniasis

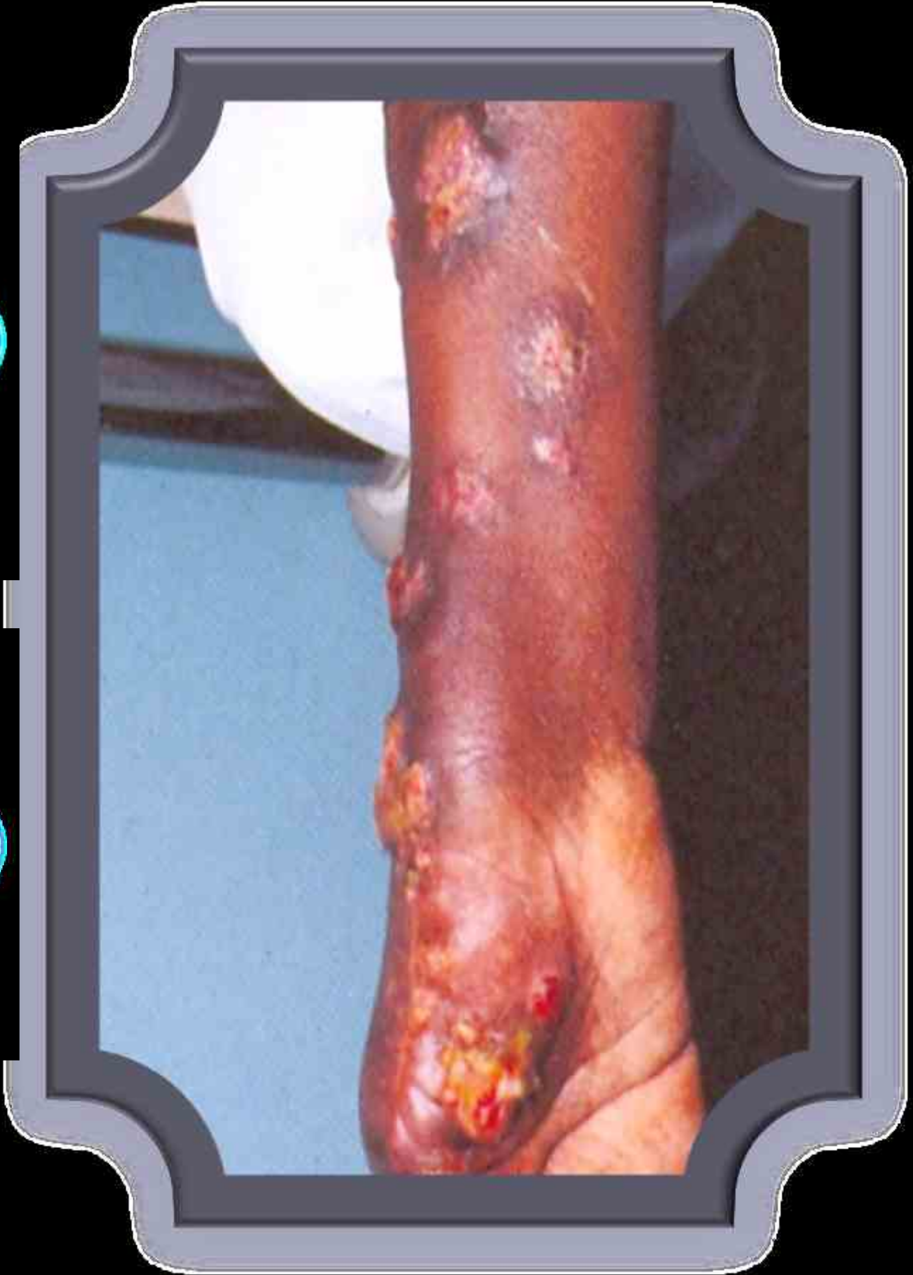
Leg: Large Ulcer with Undermined Edge



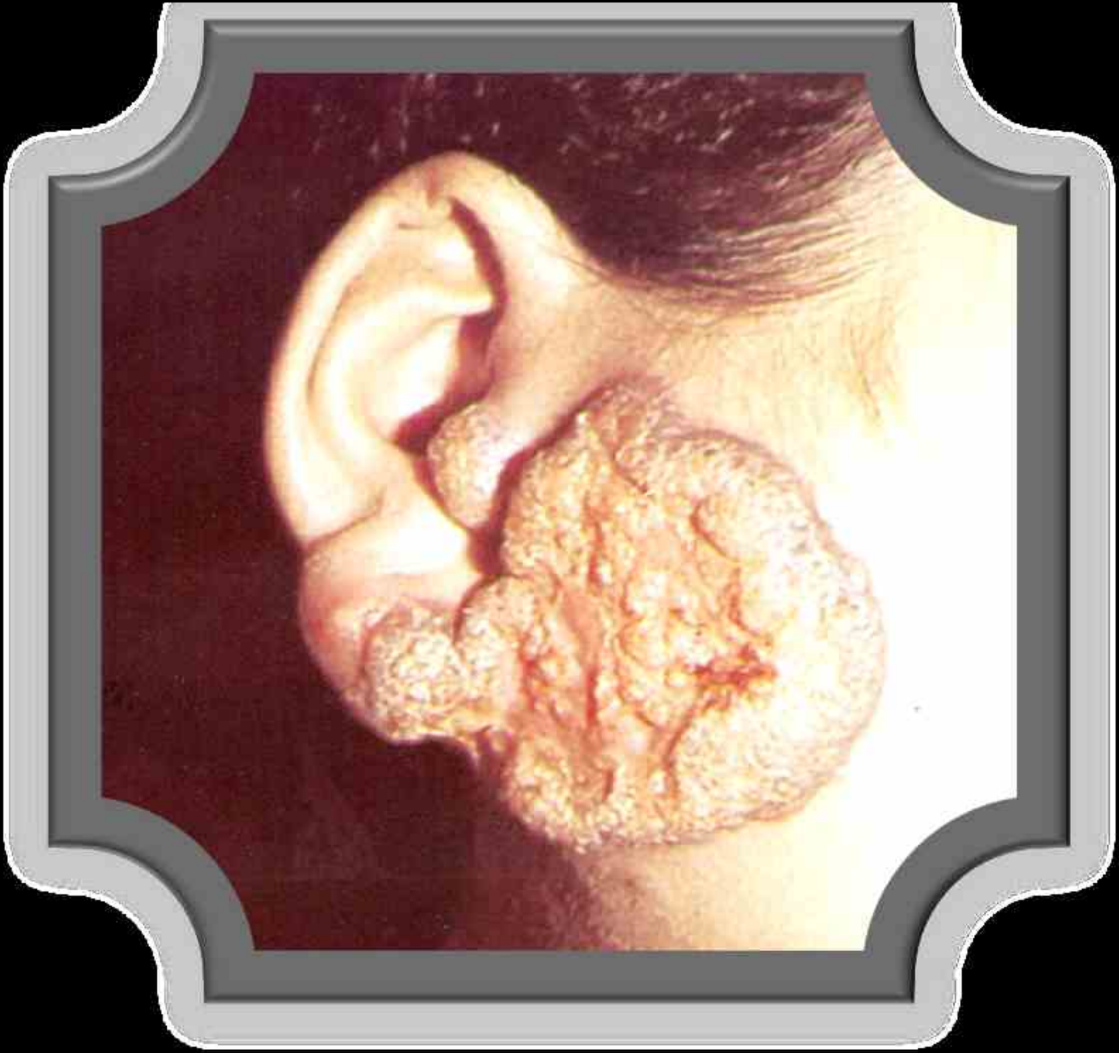
Cutaneous Leishmaniasis

Multiple large Ulcers

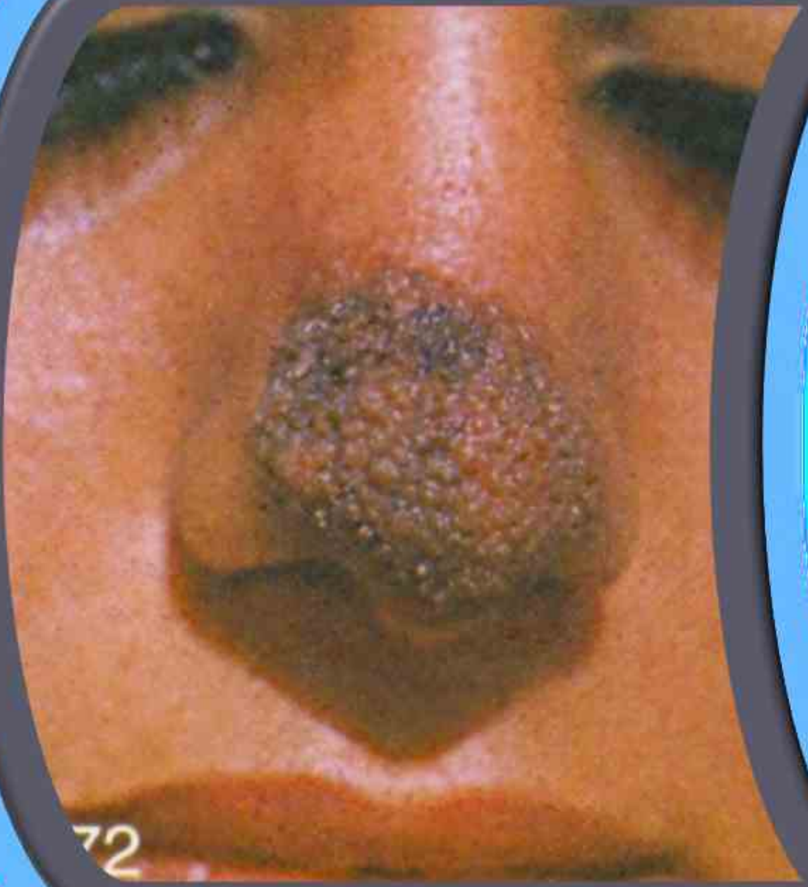
Sporotrichoid Distribution



Cutaneous Leishmaniasis
Non-ulcerated lesion on ear and lateral side of face

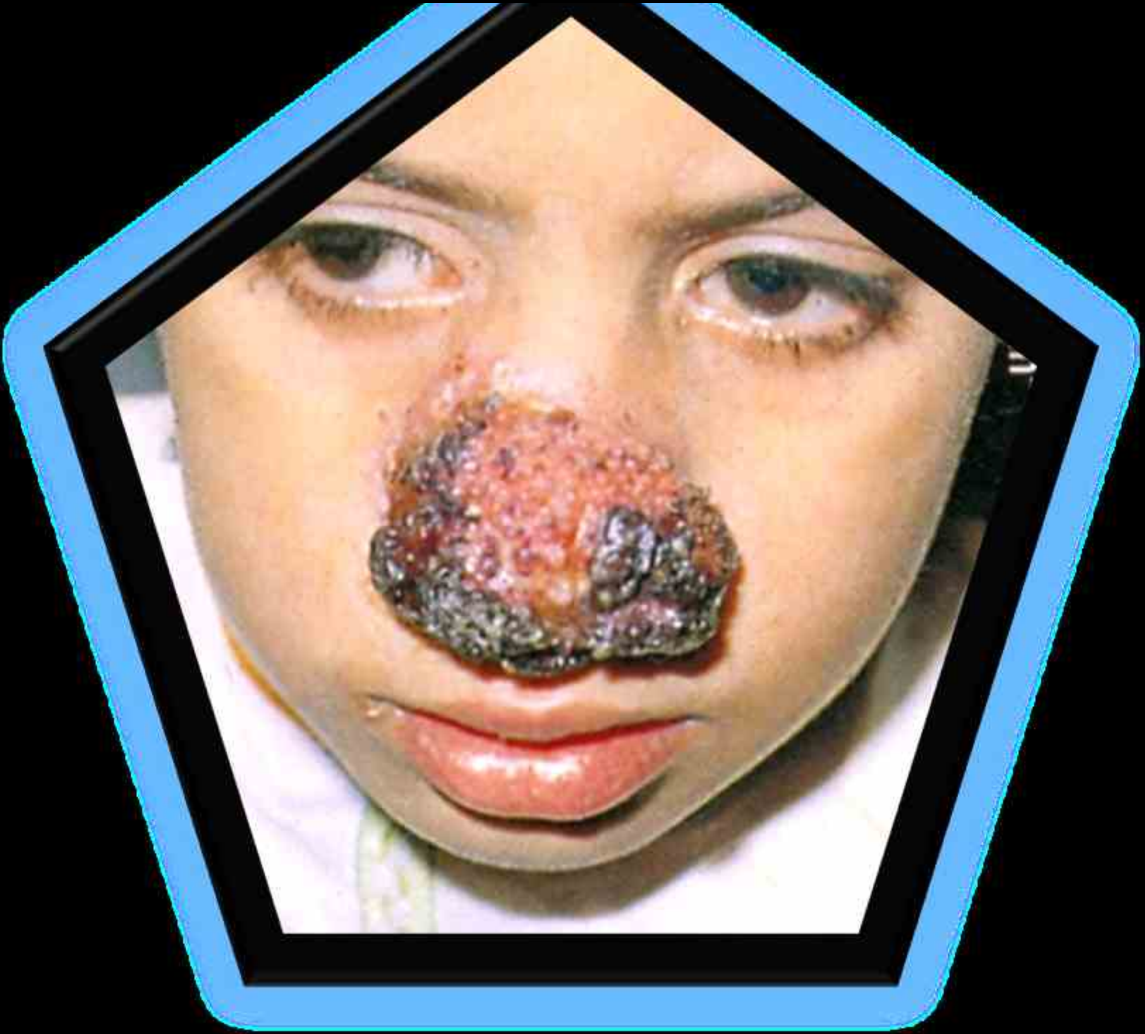


Cutaneous Leishmaniasis Non-ulcerated lesion on the nose

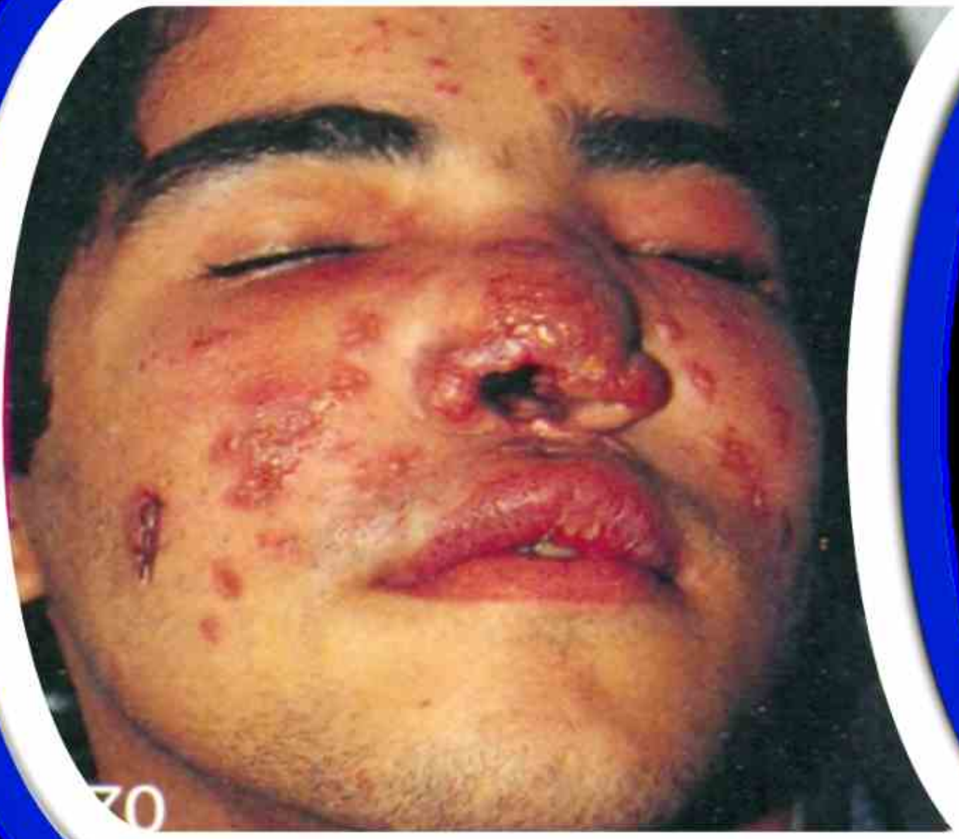


Cutaneous Leishmaniasis

Large crusted lesion on nose



Mucocutaneous Leishmaniasis



Cutaneous Leishmaniasis

B. Chronic Cutaneous Leishmaniasis: Lupoid Leishmaniasis::

- Plaque:
  - One or more.
  - Non-ulcerated.
  - Usually painless.
  - Dusky –red erythema.
  - Lasts for years.

C. Leishmaniasis Recidivans:

- Reactivation: many years after healing of old leishmaniasis lesion .
- Many plaques at or near the periphery of the scars of old lesion.

D. Diffuse Cutaneous Leishmaniasis:

- Common in Africa, in patients with immune deficiency.
- Lesions: wide spread nodules or plaque-like lesions.
- No tendency to ulceration.
- Negative Leishmanin test (Anergy).

Cut. Leishmanisis
Investigations

Cutaneous Leishmaniasis

Investigations:

1. Skin smear:

- *Bloodless sample from the edge of the lesion.*
- *Stained with Geimsa stain.*
- *Under the light microscopy → Extra and intracellular Amastigotes form(leishmania bodies).*

2. Culture:

- *On NNN media → Promastigotes.*

3. Biopsy:

In chronic cases with negative smears.

4. Immunological test:

a. **Leishmanin test (Montenegro): Intradermal test:**

- *Positive → Erythematous papule (infection).*
- *Negative → 1. Non-infected.*
  2. Persons with disseminated cutaneous leishmaniasis (DCL).

b. **Serum examination:** *for leishmania antibodies.*

Cutaneous Leishmaniasis

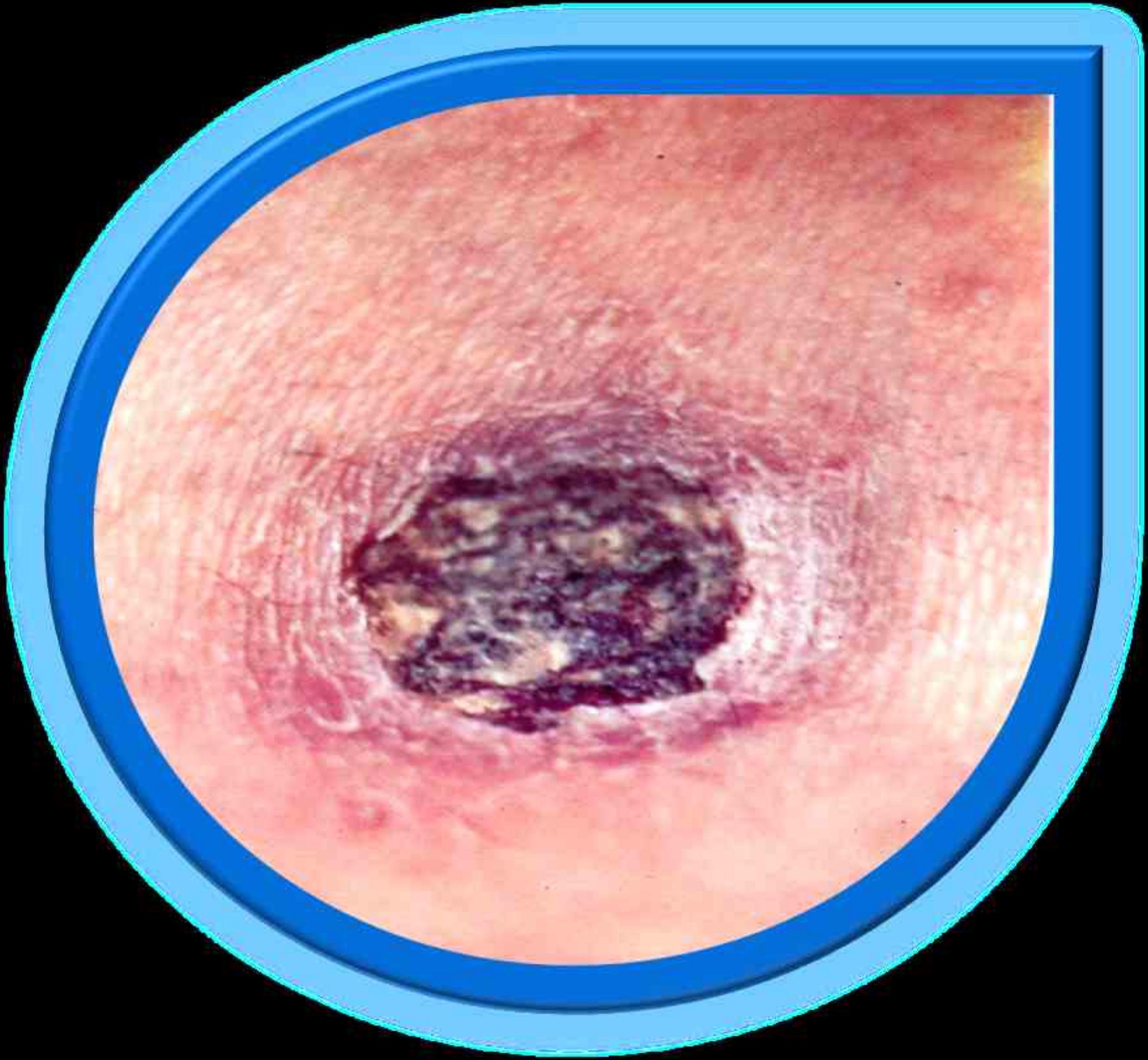
Diagnosis:

1. **History** : Patient from endemic area.
   Chronic skin lesion not respond to treatment.
2. **Clinical examination**→ characteristic lesion on exposed area.
3. **Investigations.**

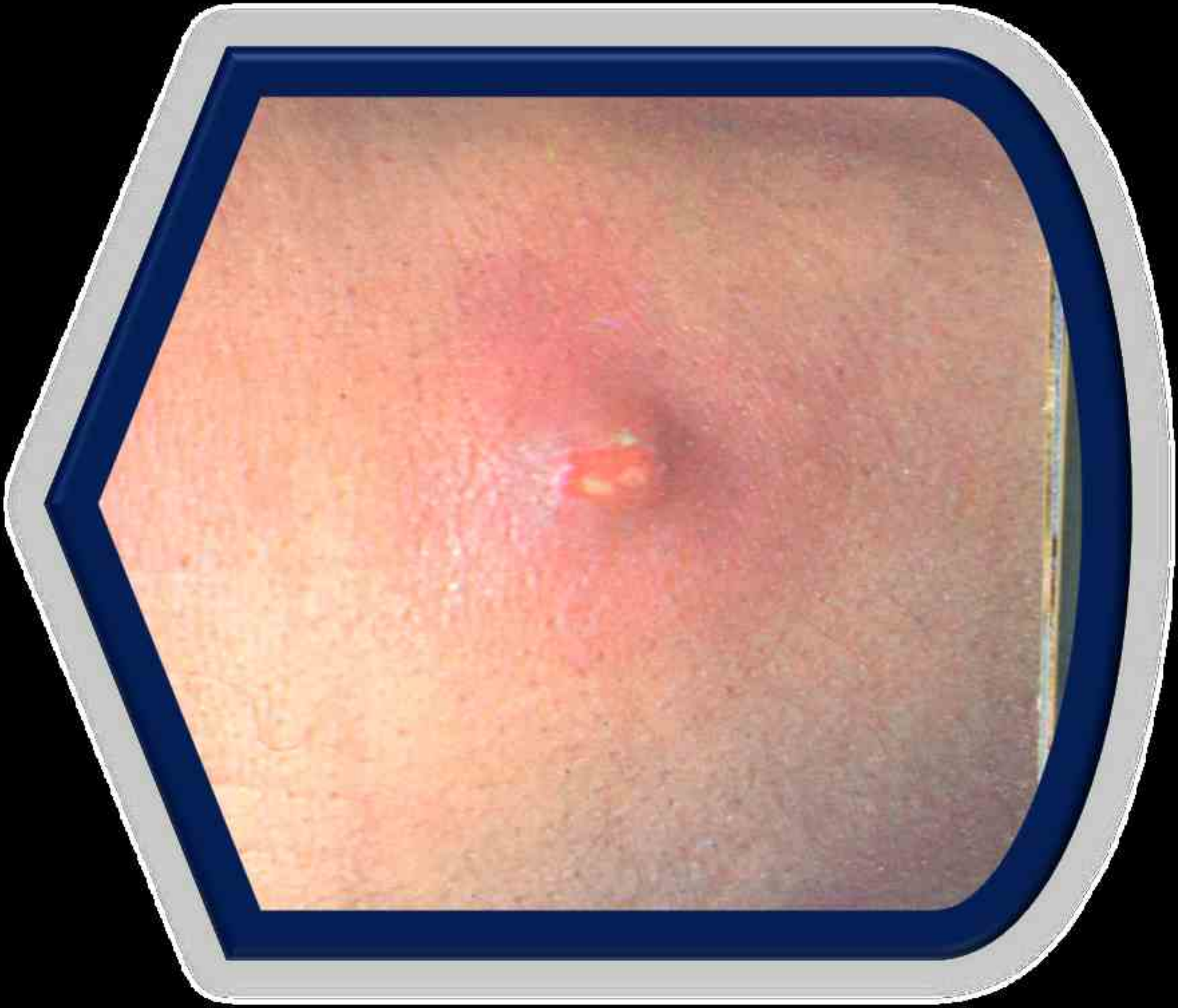
Differential diagnosis:

1. Ecthyma.
2. Furuncle.
3. Lupus vulgaris.

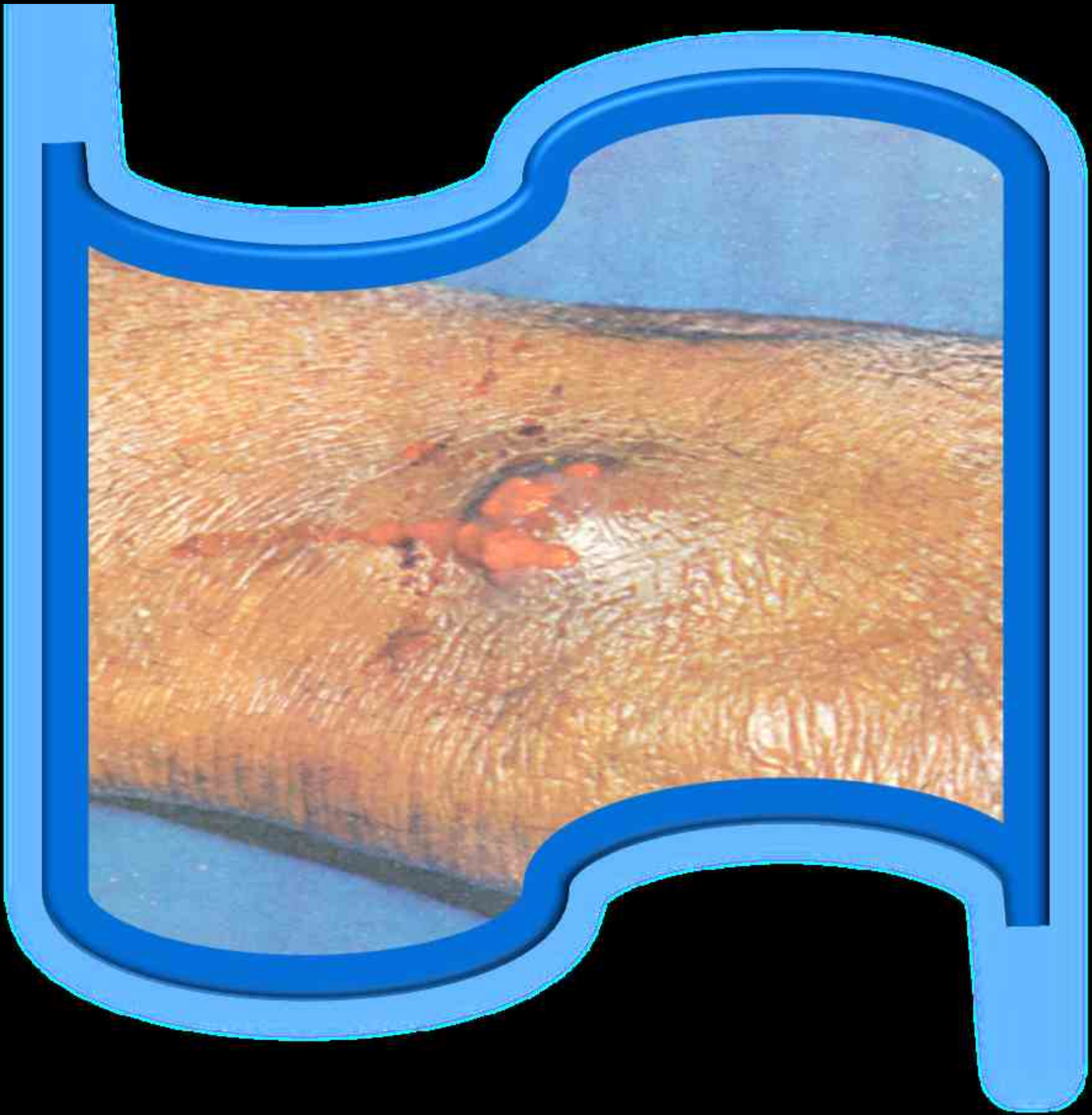
Ecthyma



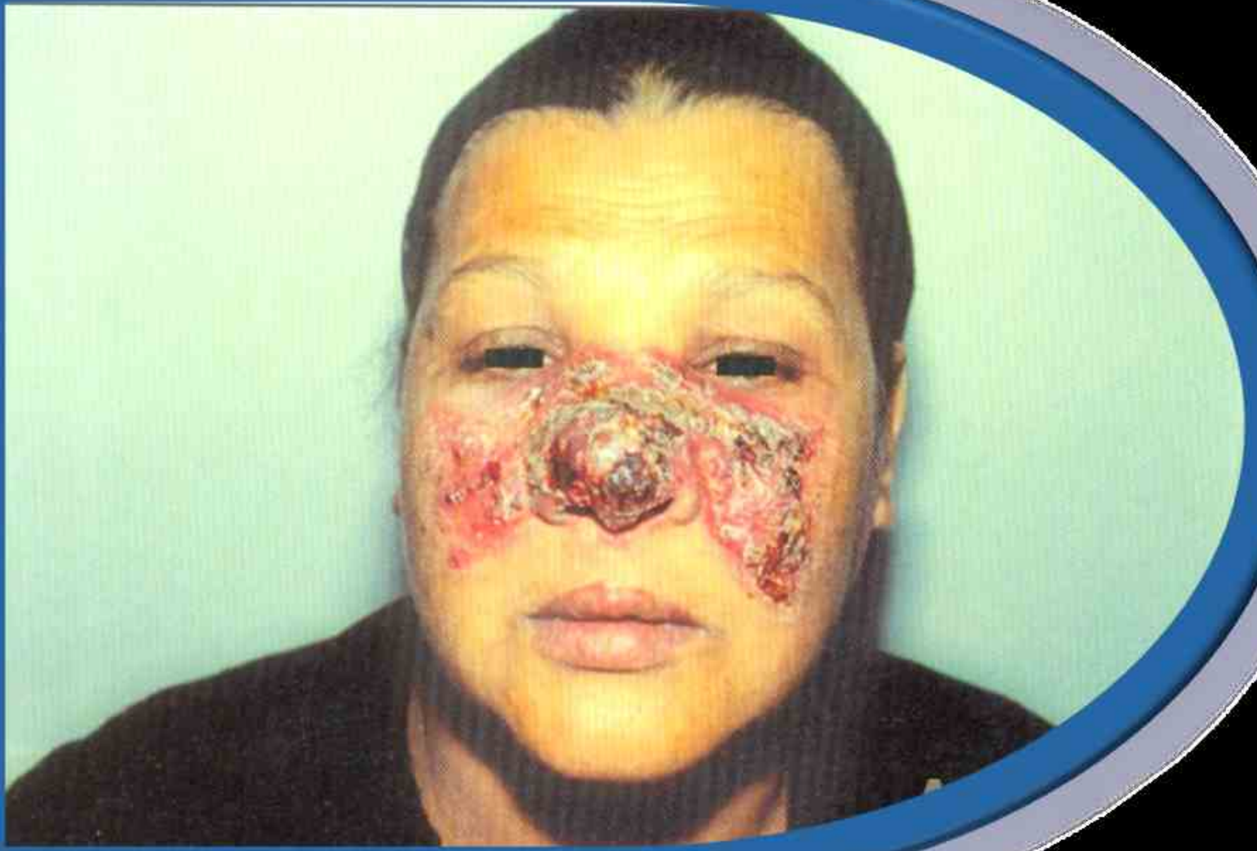
Furuncles



Carbuncle

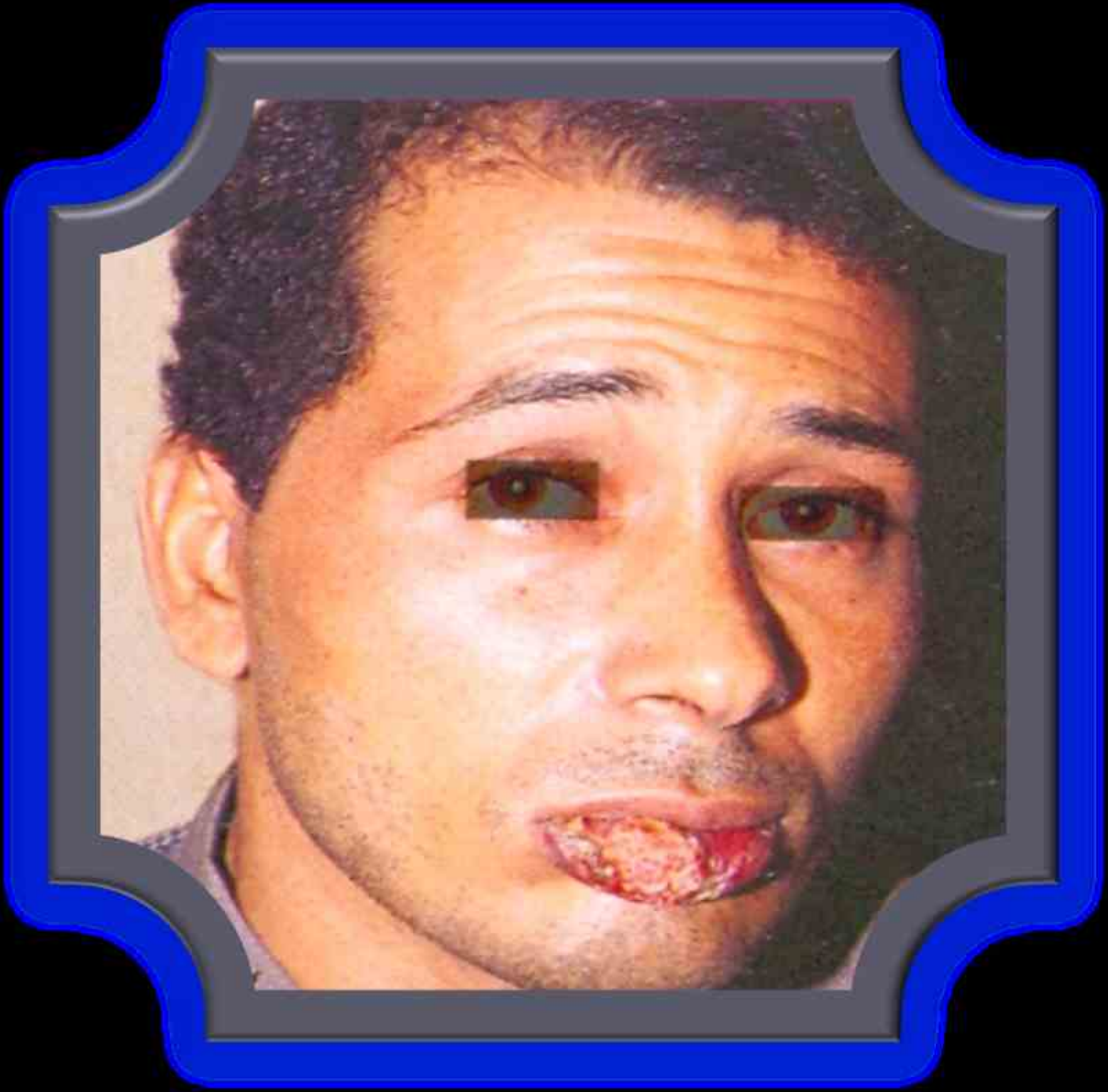


Lupus Vulgaris
Cutaneous tuberculosis

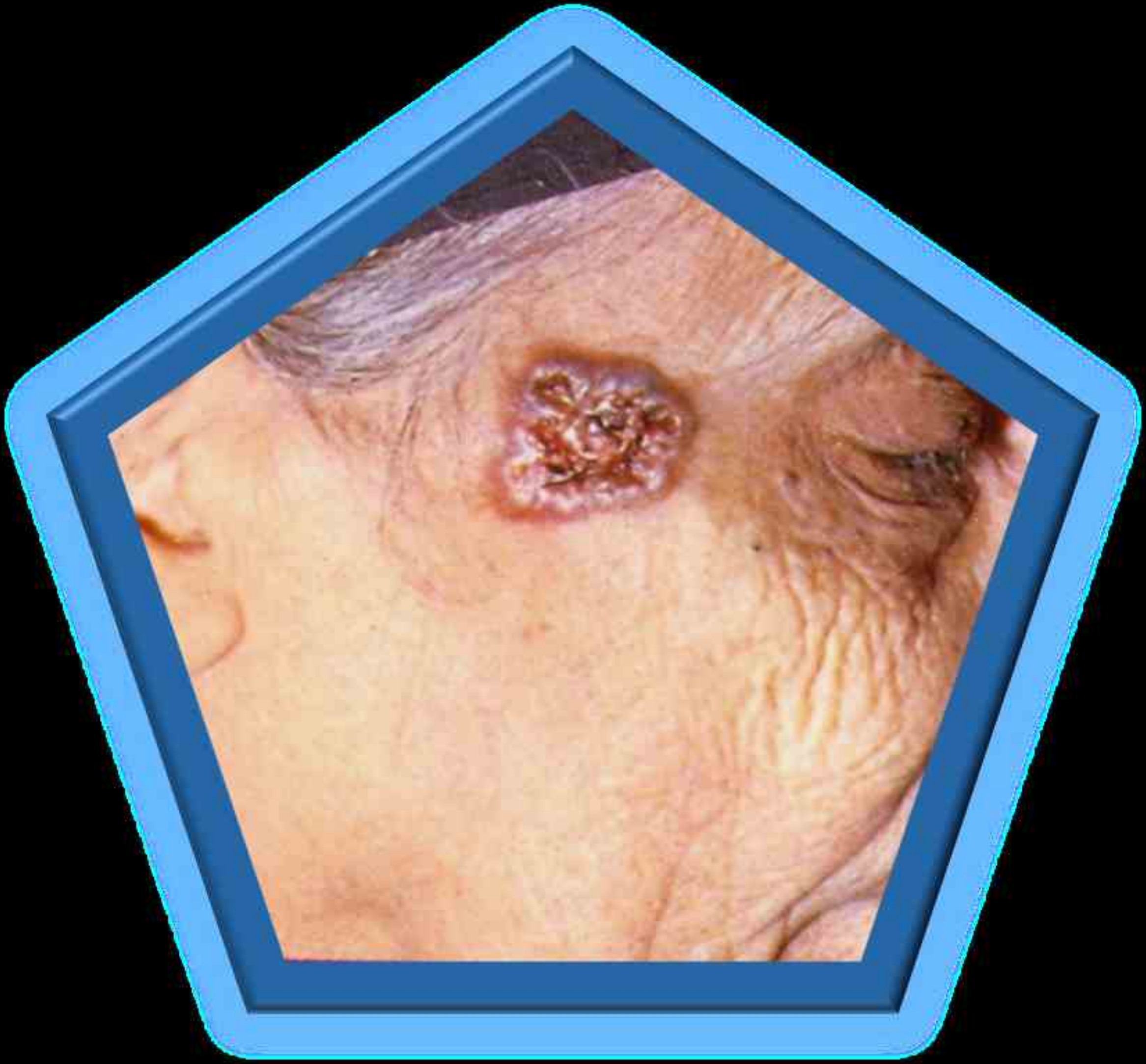


Squamous Cell Carcinoma

On exposed part of lower lip



Basal Cell Carcinoma



Cutaneous Leishmaniasis
Management

A. Prophylactic measures:

1. *Treatment of patients in endemic areas.*
2. *Control of the sandfly.*
3. *Avoiding the reservoir hosts (rodents & dogs) of infection.*
4. *Improve environmental sanitary conditions.*

B. Local measures:

1. *Excision of single lesion.*
2. *Cryotherapy with carbon dioxide snow, liquid nitrogen.*
3. *Intralesional injection of antimonial drugs.*
4. *Topical antibiotic for secondary infection.*

Cutaneous Leishmaniasis
Management Continue…

C. Systemic treatment:

1. Pentavalent antimonial drugs:

❖ Sodium Stibogloconate: Pentostam®:

Dose: 10-15 mg/kg/day intramuscularly for 2 – 3 weeks.

Or :

Intralesional: 1 – 3 ml per lesion/ once or twice/ week.

Side effects:

1. Painful injections.
2. Hypersensitivity (anaphylaxis)
3. ECG changes.
4. Hemolytic anemia.
5. Hepato-renal damage.

Contraindications:

1. Allergy.
2. Pregnancy and lactation.
3. Cardiac patients.
4. Hepato-renal diseases.

Cutaneoous Leishmaniasis

Management Continue…

2. Other drugs:

1. Rifampicine: Rifadin®
   600 mg orally/day for 4 – 6 weeks.
2. Metronidazole: Flagyl®
   250 mg orally/3/day for 10 days.
3. ketoconazole: Nizoral®
   200 mg orally/ 3/day for 4 – 12 weeks.
4. Itraconazol: Sporanox®
   200 mg orally/ twice/ daily for 4 weeks.

CUTANEOUS

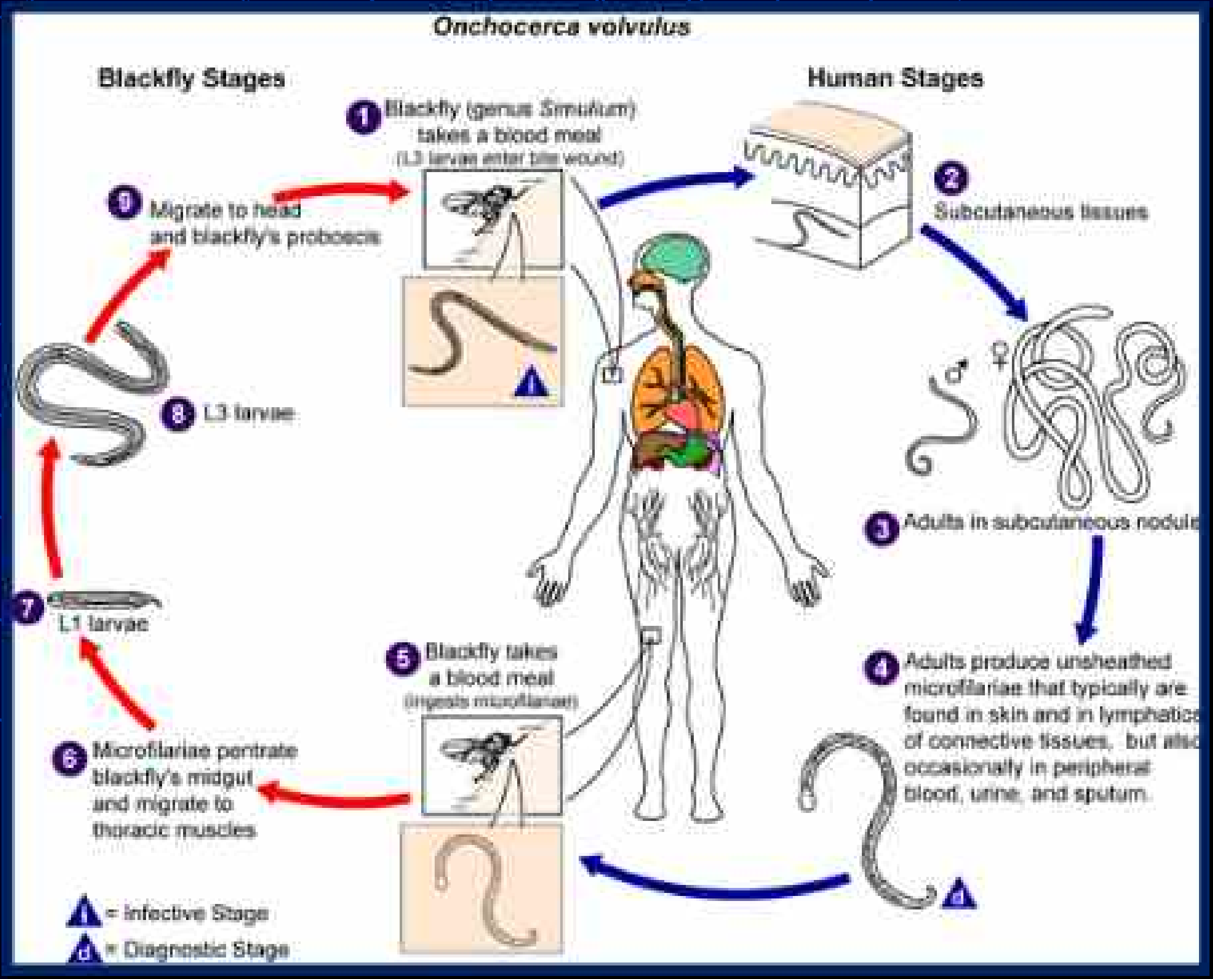
ONCHOCERCIASIS

CUTANEOUS ONCHOCERCIASIS

Onchocerciasis or “River blindness” is:

- *A filarial infestation, affects cutaneous and ocular tissue.*
- *Caused by Onchocerca volvulus.*
- *Transmitted by a bite of a black fly from the genus “Simulium”. which its larvae & pupae thrive well near rivers and waterfalls.*
- *Affects about 40 million people, particularly in Africa and Central and South America.*
- *The disease is endemic in some areas of Yemen, where it called the “Sowda” or “Assowda” .*

Onchocerca Volvulus
Life Cycle



Onchocerca volvulus
Blackfly Stages
Human Stages
1 Blackfly (genus *Simulium*) takes a blood meal (L3 larvae enter bite wound)
2 Subcutaneous tissues
3 Adults in subcutaneous nodule
♂
♀
4 Adults produce unsheathed microfilariae that typically are found in skin and in lymphatics of connective tissues, but also occasionally in peripheral blood, urine, and sputum.
5 Blackfly takes a blood meal (ingests microfilariae)
6 Microfilariae pentrate blackfly's midgut and migrate to thoracic muscles
7 L1 larvae
8 L3 larvae
9 Migrate to head and blackfly's proboscis
i
d
i = Infective Stage
d = Diagnostic Stage

Cut. Onchocerciasis

Mode of infection

- *Black fly bite infected human → **take O. V. microfilariae, which grow in the thoracic muscles of the fly**,* where they develop into
- ***the infective e stage larvae within 6 - 12 days.***
- *The fly introduce the larvae into the skin of new host → they grow into adult male & female worm in about year within a nodule in the subcutaneous tissue.*
- *The female producing microfilariae which migrate into neighboring tissue including the eye (if nodule on the head).*
- *Microfilariae causing little inflammation → if degenerate →*

trigger an inflammatory reaction → intense pruritis.

Cut. Onchocerciasis

Clinical Picture

- **Standardized grade system:**

1. *Acute papular onchodermatitis.*
2. *Chronic papular onchodermatitis*
3. *Lichenified onchodermatitis.*
4. *Onchocercal atrophy.*
5. *Depigmentation.*

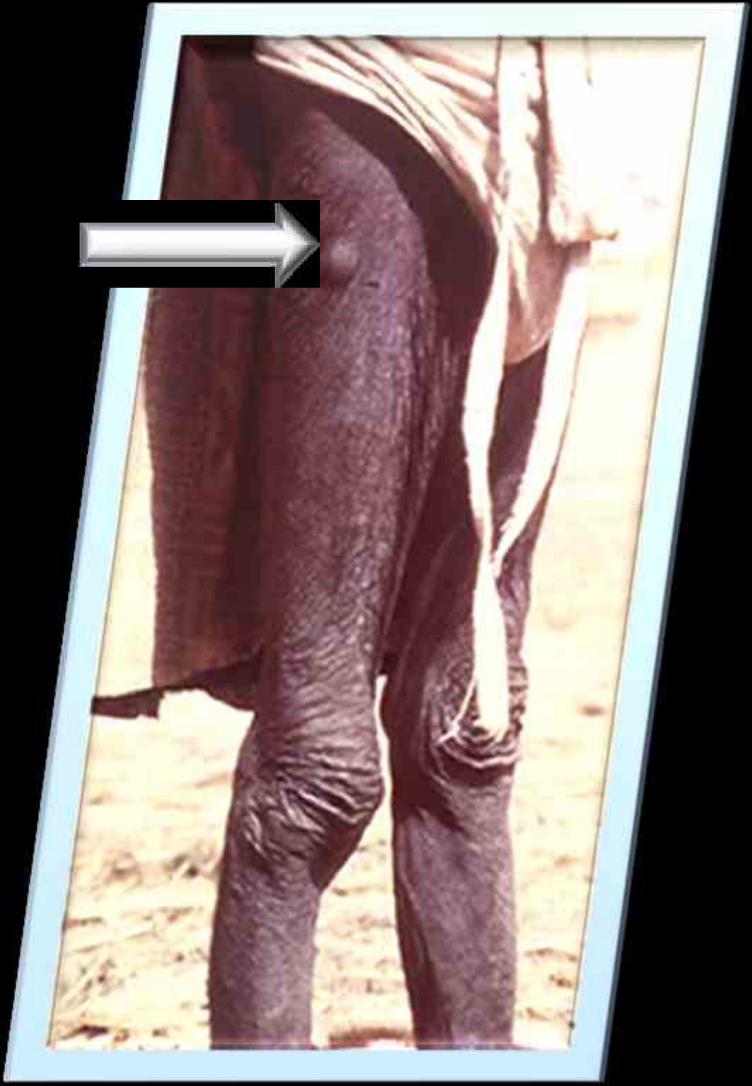
Cut. Onchocerciasis
Clinical Picture

- *Patient present first with subcutaneous nodule usually over bony prominence in the upper or lower part of the body.*
- *The nodule contains the adult worms surrounded by fibrous tissue (onchocercomata).*

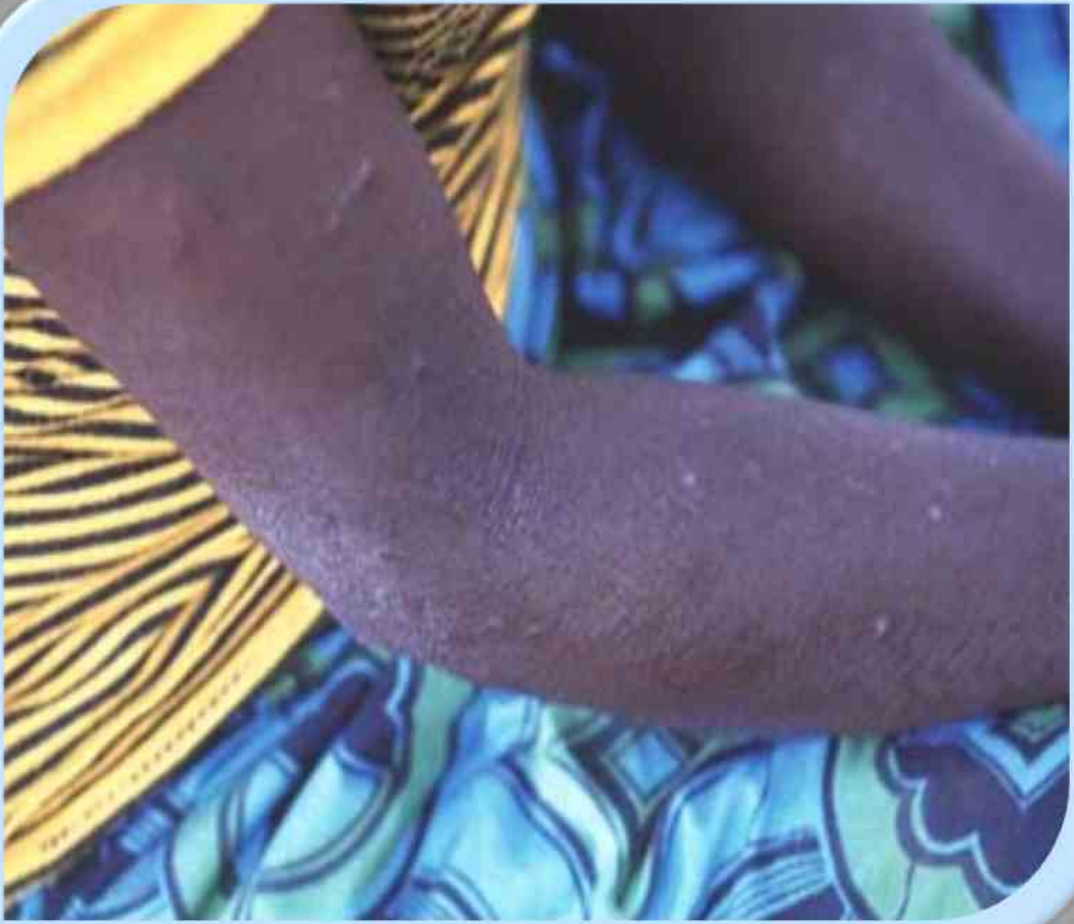
- *Acute O. → Intermittent intensely pruritic dermatitis (papules around microfilariae), abscesses may be formed if patient treated by diethyle carbamazepine .*
- *Chronic O. → Skin then become thickened, wrinkled, lichenified and hyperpigmented → Lizard or elephant skin.*

- *Late stage → loss of pigment with sparing the skin around hair follicles and atrophy → Leopard skin (more on the shins).*
- *Chronic lymphatic obstruction and involvement of the inguinal lymph nodes → Hanging groin and elephantiasis of genitalia.*

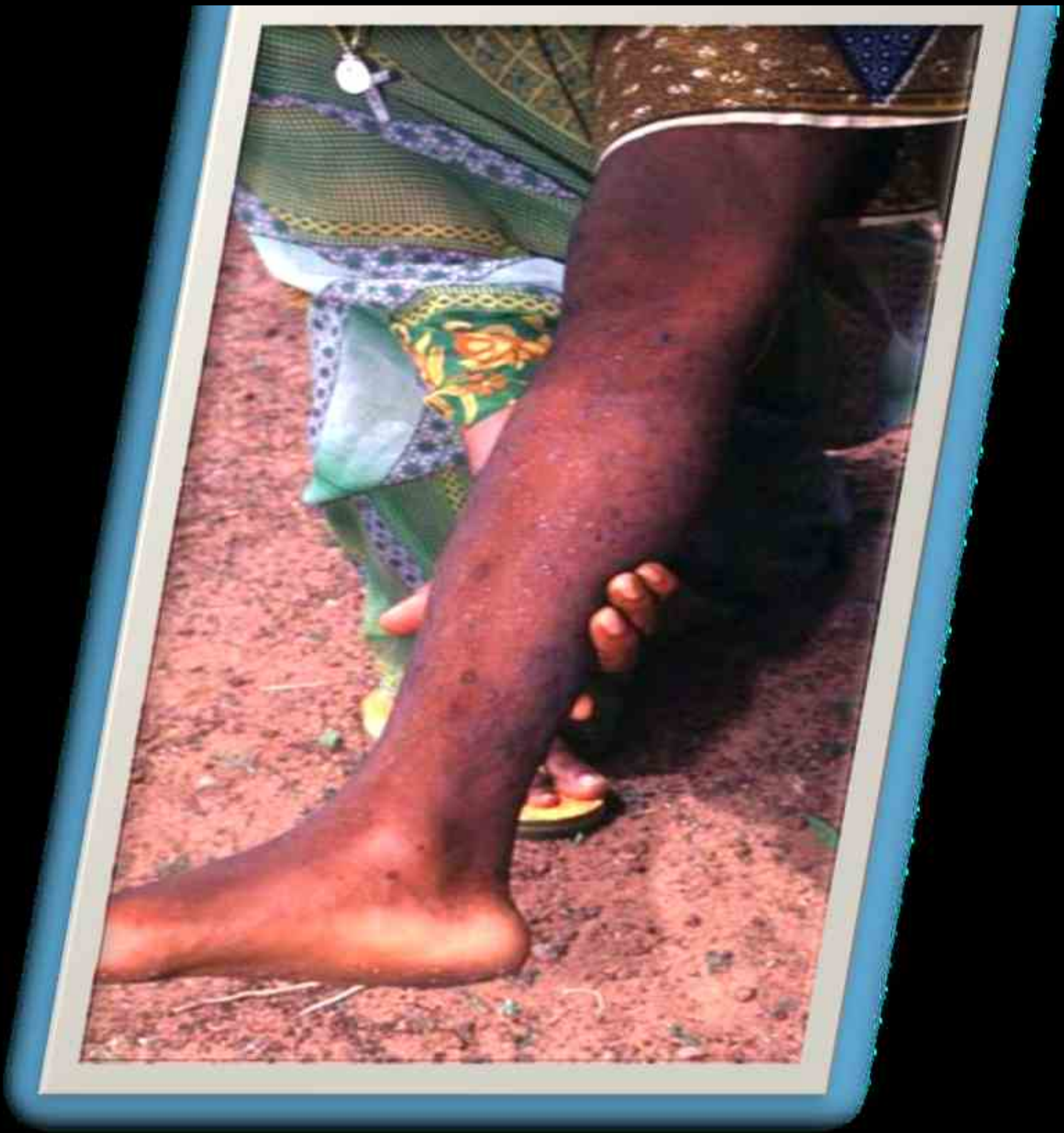
Onchocerciasis Cut.
Subcutaneous nodule



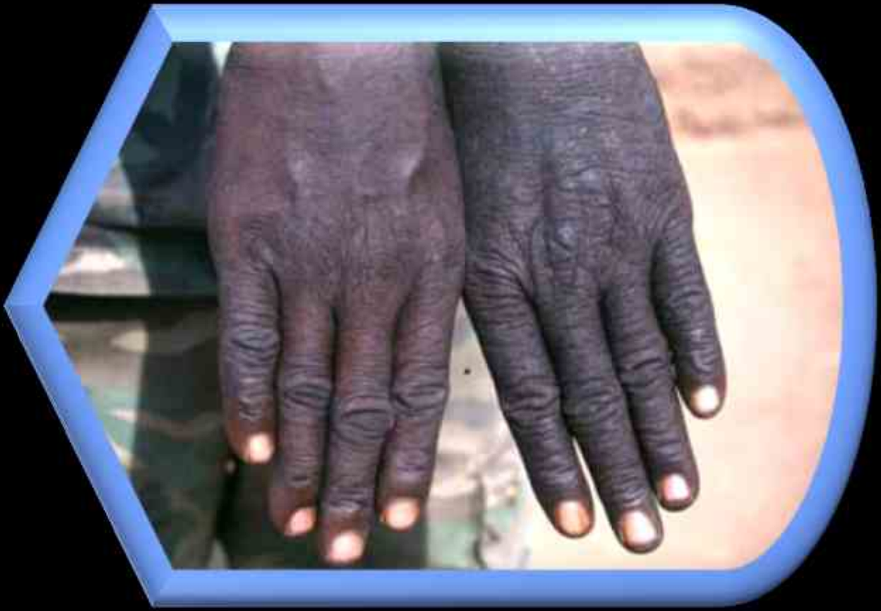
Cut. Onchocerciasis
Acute Dermatitis



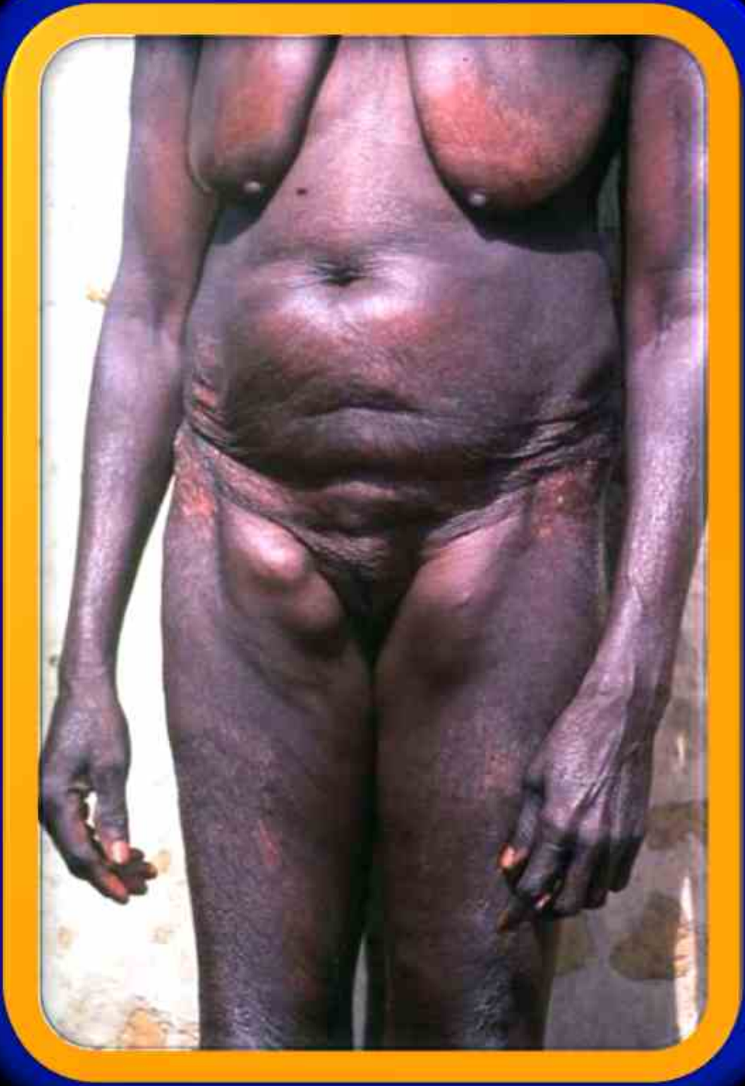
Cut. Onchocerciasis
Early papular lesions



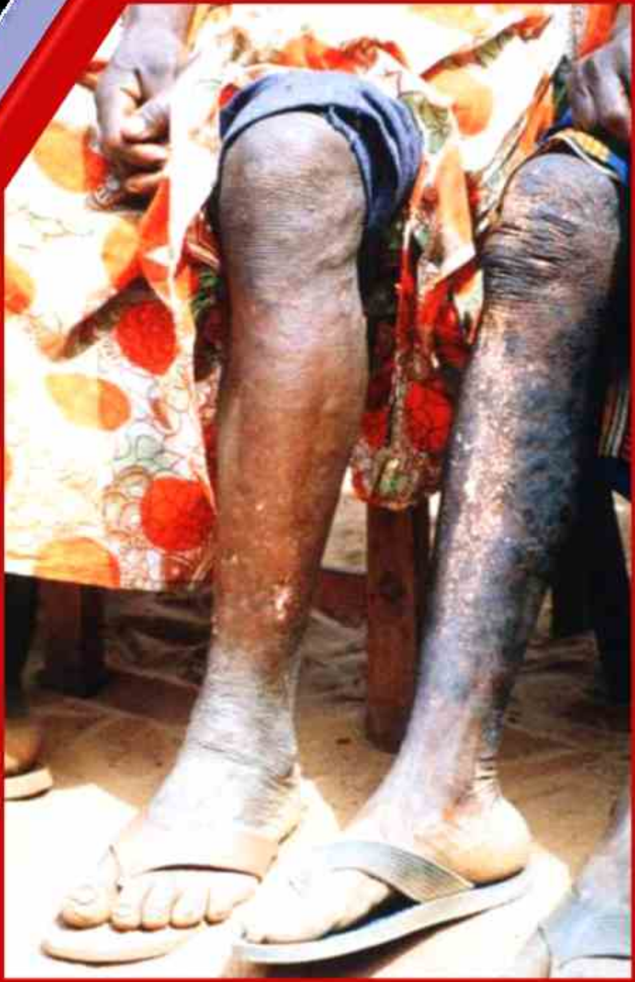
Cut. Onchocerciasis
Hyperpigmentation (Assowda)



Cut. Onchocerciasis
Inguinal Lymphadenopathy



Cut.Onchocerciasis
Lichnified skin Lesions



Ocular Onchocerciasis

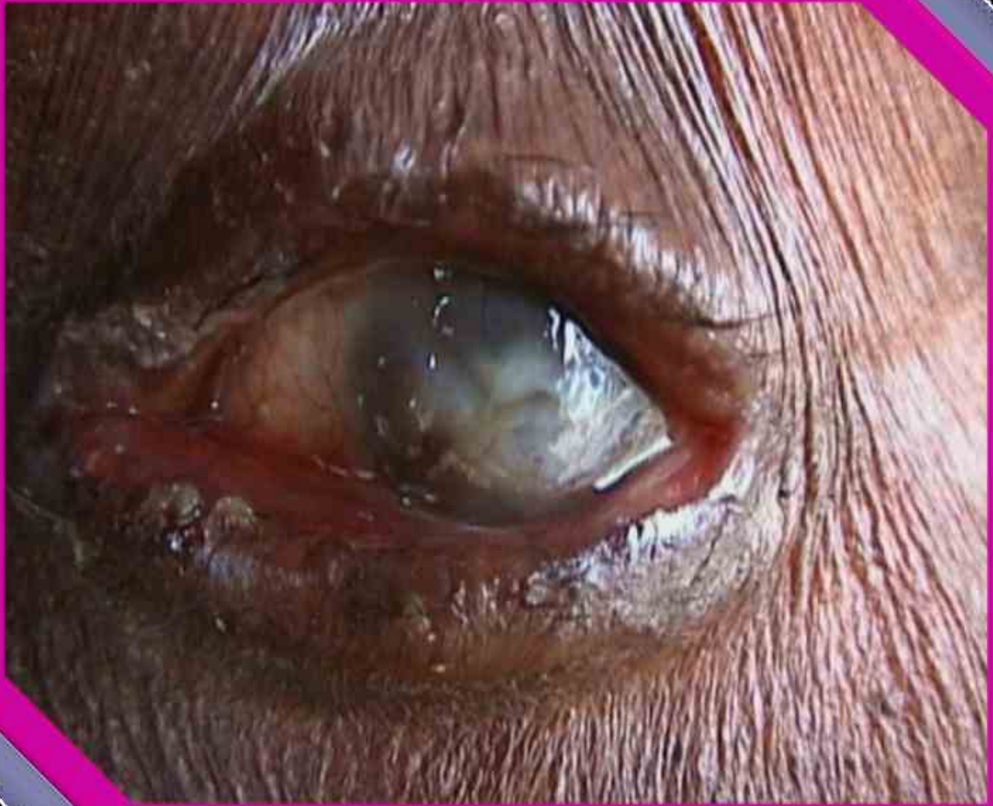
Clinical Picture

- *Microfilariae in the conjunctivae → through the cornea → anterior and posterior chambers of the eye causing:*

1. *Conjunctivitis.*
2. *Sclerosing keratitis.*
3. *Uveitis.*
4. *Chorioretinal lesion.*
5. *Optic nerve atrophy.*
6. *Glucoma.*
7. *Blindness in severe cases.*
8. *Dead microfilariae in the cornea → tissue reaction → "Snow-flake" opacities.*

Ocular Onchocerciasis

Corneal opacities



Cut. Onchocerciasis

Diagnosis

- *Clinical diagnosis is not difficult in endemic areas.*
- *Confirmation require microscopic visualization of microfilariae emerging from skin sample (snip) placed in saline.*
- *PCR.*
- *Mazzotti Reaction:*
    - *Acute hypersensitivity reaction to rapidly dying microfilariae within the skin, elicited by oral diethyl carbamazepine (DEC).*
    - *Used in the past for the diagnosis if microfilariae not seen in skin snips*

Cut. Onchocerciasis
Differential Diagnosis

1. *Early lesion:*
   - *AD, insect bite , Scabies, and allergic CD.*
2. *Chronic lesion:*
   - *Ch. Eczema with post inflammatory hyperpigmentation, leprosy, scleroderma.*
3. *Elephantiasis:*
   - *From other types of filarial infection.*

Cut. Onchocerciasis
Treatment

- Ivermectin is the drug of choice.
- Action:
  - Rapid killing of microfilariae.
  - Prevents their escape from gravid female.
  - No or few adverse reaction or Mazzoti reaction.
- Dose:
  - 150 - 200 µg/kg single oral dose.
  - Multiple doses can kill adult form as well.
- Nodulectomy.
- Preventive measures: eradication of black fly in water and treatment of patients.
- Diethyl carbamazepine and Suramin (not used), it can induce severe hypersensitivity reaction or dangerous toxic reaction.

Disorders of Sebaceous Glands

AbdelwasaeAlmekhlafi,MD

Acne Vulgaris

Acne vulgaris is a chronic inflammatory disease of the pilosebaceous units.

Pathogenesis

- Acne vulgaris is exclusively a follicular disease, with comedo formation produced by the impaction and distension of the follicles with tightly packed horny cells. Disruption of the follicular epithelium permits discharge of the follicular contents into the dermis. This, in turn, causes the formation of inflammatory papules, pustules, nodules and cysts.

Etiology Acne vulgaris is a multifactorial disease.

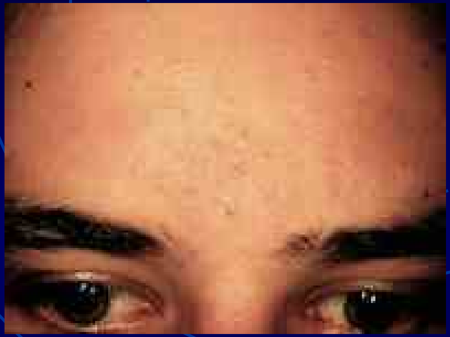
- **Genetic factors.** Several members of the same family may be affected.
- **Androgenic stimulation of sebaceous glands** with increased sebum.
- **Ductal hypercornification.** Comedones represent the retention of keratinocytes in the duct.
- **Presence of abnormal microbial flora** (*Propionibacterium*

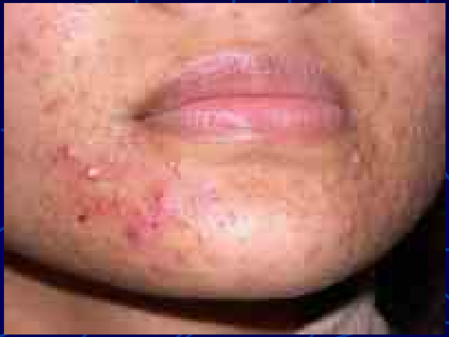
Clinical picture

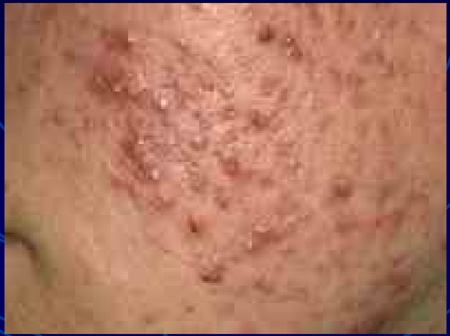
Acne vulgaris is a disease of adolescents and young adults and usually disappears by the age of 25 years. Both sexes are equally affected.

-12% of women and 3% of men will have acne until their 40s

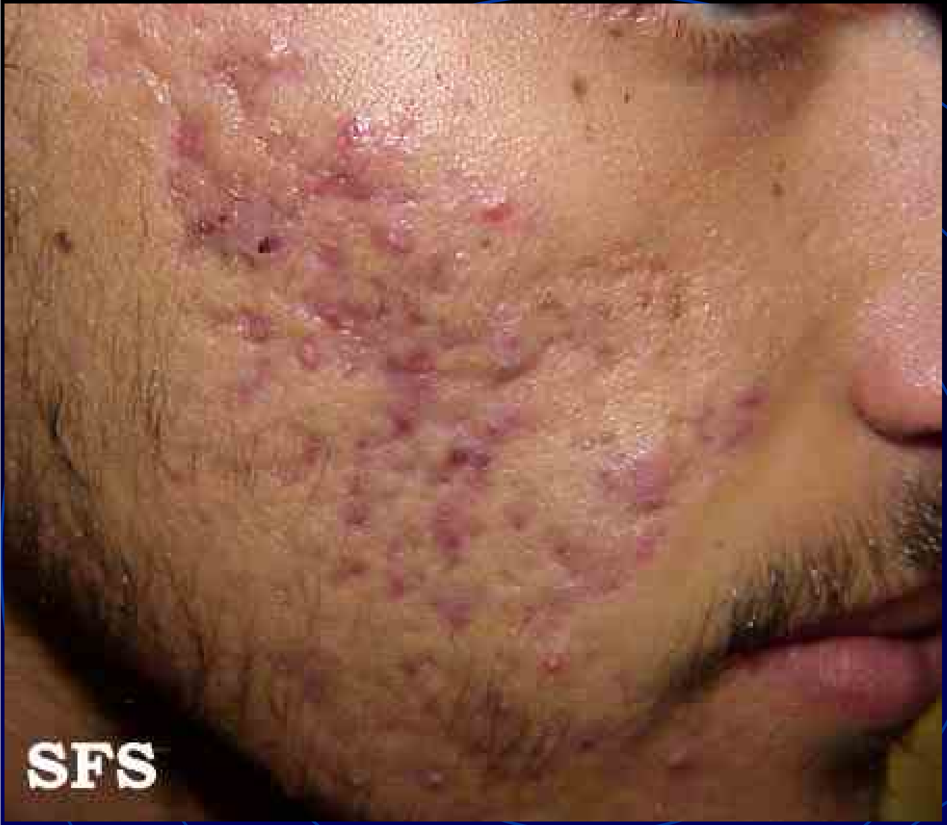
- **Sites:** It occurs predominantly on the face (99%) and, to lesser extent, on the back (60%) and chest (15%). On the face it occurs most frequently on the cheeks, and to a lesser degree on the forehead, nose and chin. Acne is a **polymorphic disease**, but one type of the following lesions may predominate
- **Comedones (blackheads)**. Comedo is the basic lesion in acne.It is a small punctate black lesion. When a comedo is extracted outside the follicular canal it appears as a yellowish worm-like structure with an upper black end (the black color is due to melanin not dirt).
- **Papules.** Small red papules.
- **Pustules.** Conical pustules.
- **Nodules and cysts. Nodules**, cysts, grouped comedones and scarring are features of acne conglobata which is a chronic disorder that may last up to 40-50 years of age.
- **Post-inflammatory lesions.** As erythema, pigmentation and

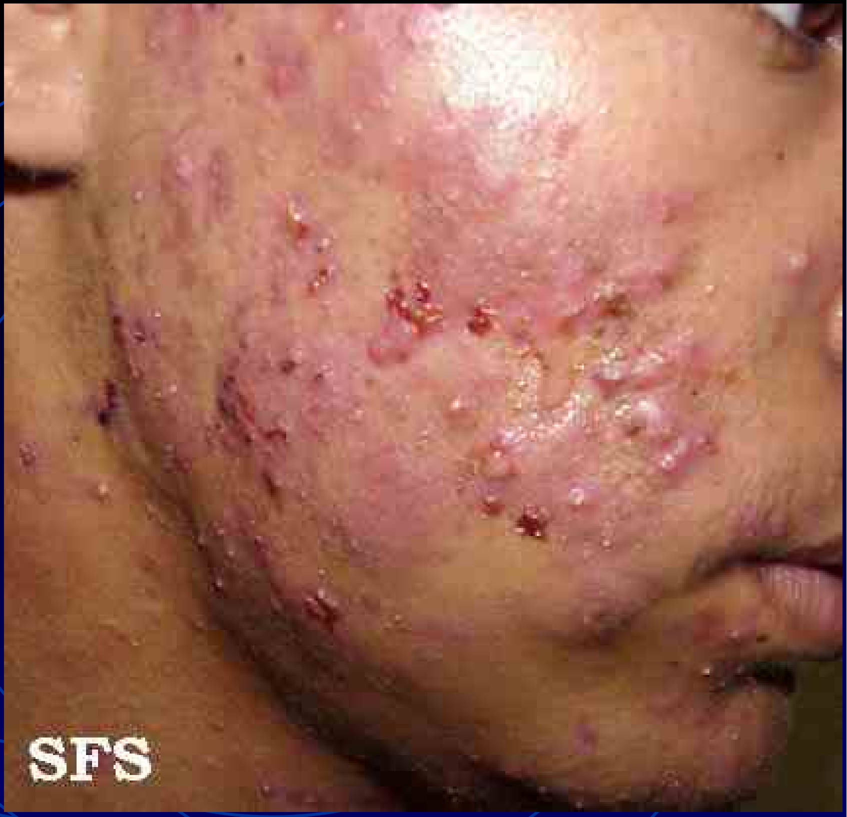


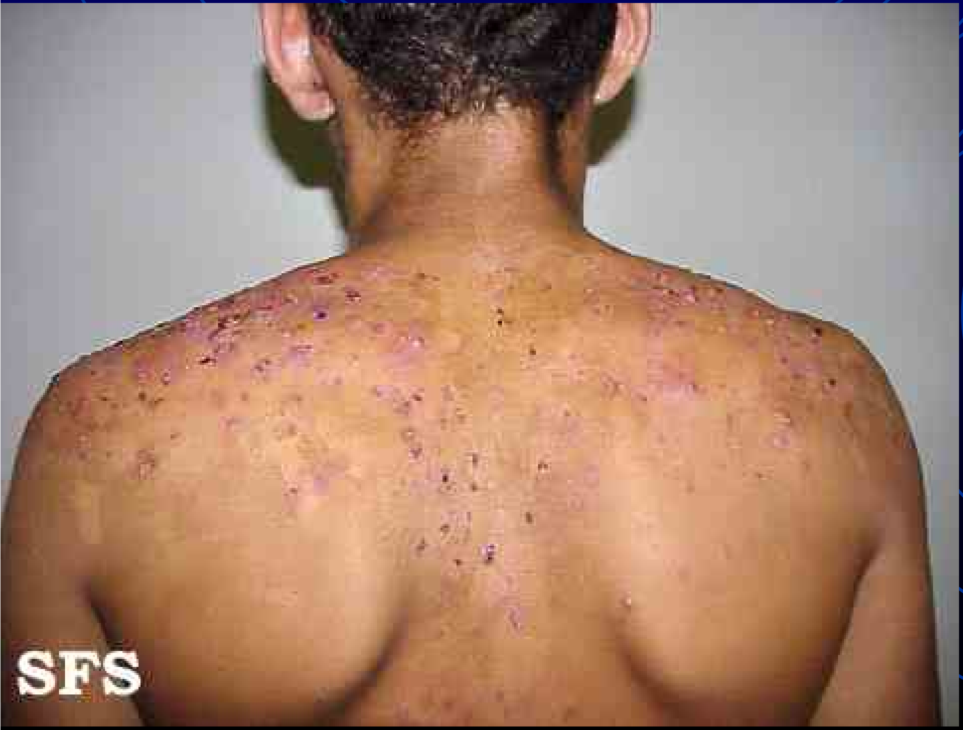


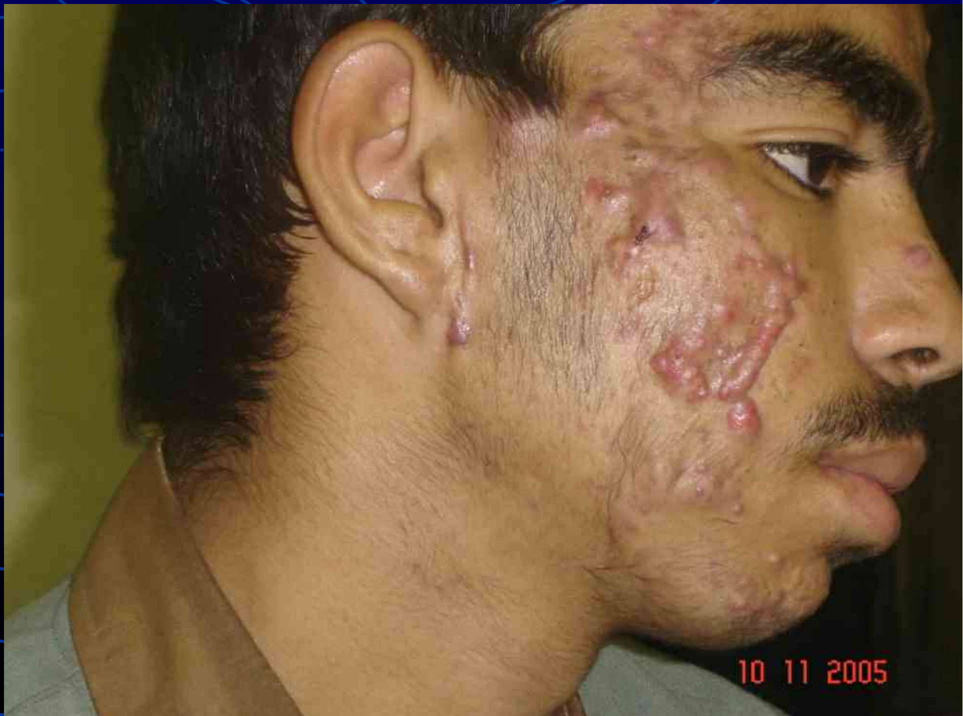


AV









Factors which may aggravate acne

- **Menstruation.** About 70% of women complain of a flare 2-7 days premenstrually.
- **Stress.** It is unlikely that stress alone induces acne lesions. However, acne itself induces stress, and “picking” of the spots will aggravate the appearance. This is particularly obvious in young females who present with acne excoriée.
- **Drugs:** bromides, steroids →acne lesions (Acne form eruption)
- **Diet:** some fats and high carbohydrate may make acne worse.
- **Sweating**. Up to 15% of acne patients notice that

- D.D.

  -**Acne form eruption,** sudden oncet, no comedones, resolved with elemination of the cause.

  -**Rosacea,** middle age, associated with redness, telenjectasia, papules , pustules but not comedones

  - **perioral dermatitis.**

Complications:

- Secondary infection, - scaring, - atrophic pits and hypertrophic scar and keloid formaton

Treatment

A- Topical

- - Topical antibiotics e.g. erythromycin, clindamycin.
- - Benzoyl peroxide 5%, 2.5%. -Retinoic acid(teritonoin) RetinA (0.025 -0.05%) -Azaleic acid (20%). - Adapalen, intralesion st for cystic lesions.

B- Systemic

- *1. Antibiotics.* They are the most important and should be administered for long periods at least 6 months. The mode of action of the antibiotics is not to reduce the number of bacteria, but they affect their function. The commonly used antibiotics are :
- **Tetracycline.** It is the safest and cheapest and used as 250 to 500 mg one to four times daily (on empty stomach) initially with gradual reduction of the dose, depending on clinical response.
- **Erythromycin.** For those who cannot take tetracycline or in pregnant women.
- **Others** e.g. clindamycin, cotrimoxazole.
- *2. Antiandrogens.* Antiandrogens (cyproterone acetate) are used only in adult females.
- *3. Systemic retinoids (isotretinoin).* Used only in severe cases,
- *4. Systemic steroids.* Used only in severe cases.

Treatment of post-acne scars

- Chemical peeling. - Dermabrasion. - Laser skin resurfacing.

Acne Rosacea

chronic inflammatory condition located at the flush areas of the face (nose,checks, chin)

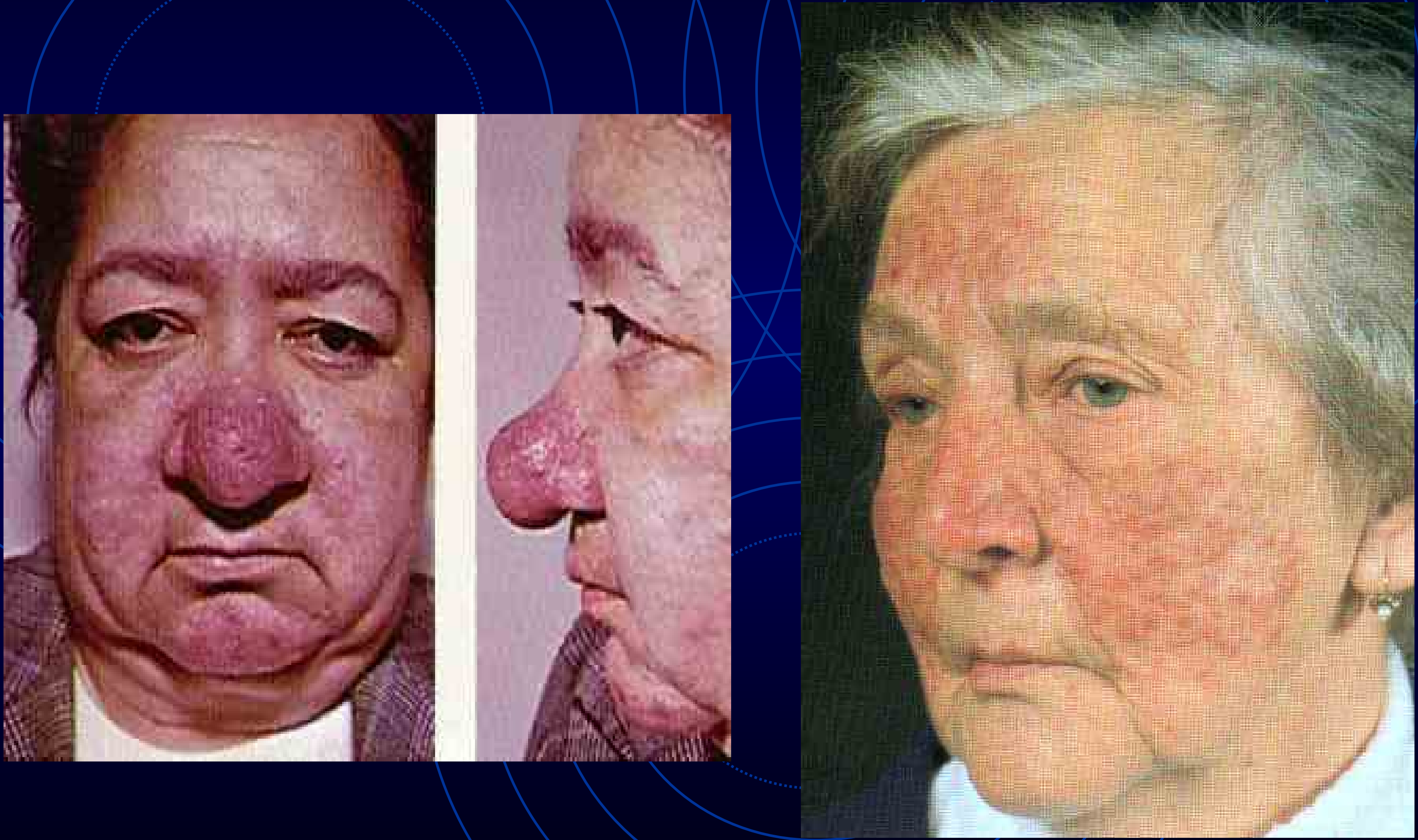
- More common in women 30s-50s (later than acne. V)
- Begins in midll age
- Central face involveed
- Erythema, edema, papules, pustule, telengictasias, Rhinophymma (dermal and sebaceous gland hyperplasia of the nose), patients can have ocular rosacea (Kiratitis, Blepharitis, Conjunctivitis)
- No comedones , not related to androgens (unlike acne.V)
- Associated with flushing
- **Trigger factors** (alcohol, heat, hot beverages, hot spicy foods, sun light, and immotional stress)
- **Multifactorial** -Vasomotor lability -Flourinated corticosteroids
- Demodex folliculorum

Treatment:

-Antibiotics: -Topical or systemic: Tetracycline – metronidazole

-Flushing triger avoidance - Topical steroid avoidance

-Surgical correction of phymatous changes



- **D.D. of Red face**

-Erysepilas - Rosacea -SLE - seb. Der -Photo der -Contact der – Urticaria

Side effects of Retinoids:

-Chelitis,epistaxis,erythema

-Teratogenic

-hepatotoxicity, hyperlipidemia

Contra indications:

- pregnancy, lactation, liver diseases, hyperlipidemia

Allergic Skin diseases

Abdelwasa Almekhlafi,MD

Eczema/Dermatitis

Eczema is an inflammatory skin reaction characterized histologically by spongiosis and clinically by itching, redness, scaling and papulovesicular eruption. The terms eczema and dermatitis are generally regarded as synonymous.

- Eczema/dermatitis may be presented as acute, subacute or chronic. In the acute eczema erythema, edema, oozing and papulovesicular eruptions are the presenting features. In the subacute eczema, the lesion shows less erythema, edema and oozing with scaling and crusting. In the chronic eczema, the lesion shows thickening and lichenification.

Varieties of eczema/dermatitis

- Contact dermatitis.
- Atopic dermatitis.
- Infective dermatitis.
- Seborrhoeic dermatitis.
- Discoid eczema.
- Pompholyx.
- Gravitational eczema.
- Pityriasis alba.

Contact dermatitis

A-Irritant contact dermatitis

- Irritant dermatitis is an inflammatory reaction in the skin resulting from exposure to a substance that causes an eruption in most people who come in contact with it if a sufficiently high concentration of the substance is used. No previous exposure to the substance is necessary, and the effect is evident within minutes or a few hours at most.

B-Allergic contact dermatitis

- Allergic contact dermatitis is an acquired sensitivity to various substances that produce inflammatory reaction in only those who have been previously exposed to the allergen (sensitizer) i.e. does not occur from the first exposure. It results from a specific acquired hypersensitivity of the delayed type i.e. cell-mediated immunity. Persons may be exposed to the allergens for years before developing dermatitis.

Clinical picture

- The clinical pictures of irritant and allergic contact dermatitis are more or less the same. The primary signs in contact dermatitis are erythema, swelling, papules and papulovesicles. If contact dermatitis persists, it may be due to continued or repeated exposure to the allergen or irritant. It becomes dry, scaly and lichenification and fissuring may develop later. The site and morphology of the lesion may point to the cause.

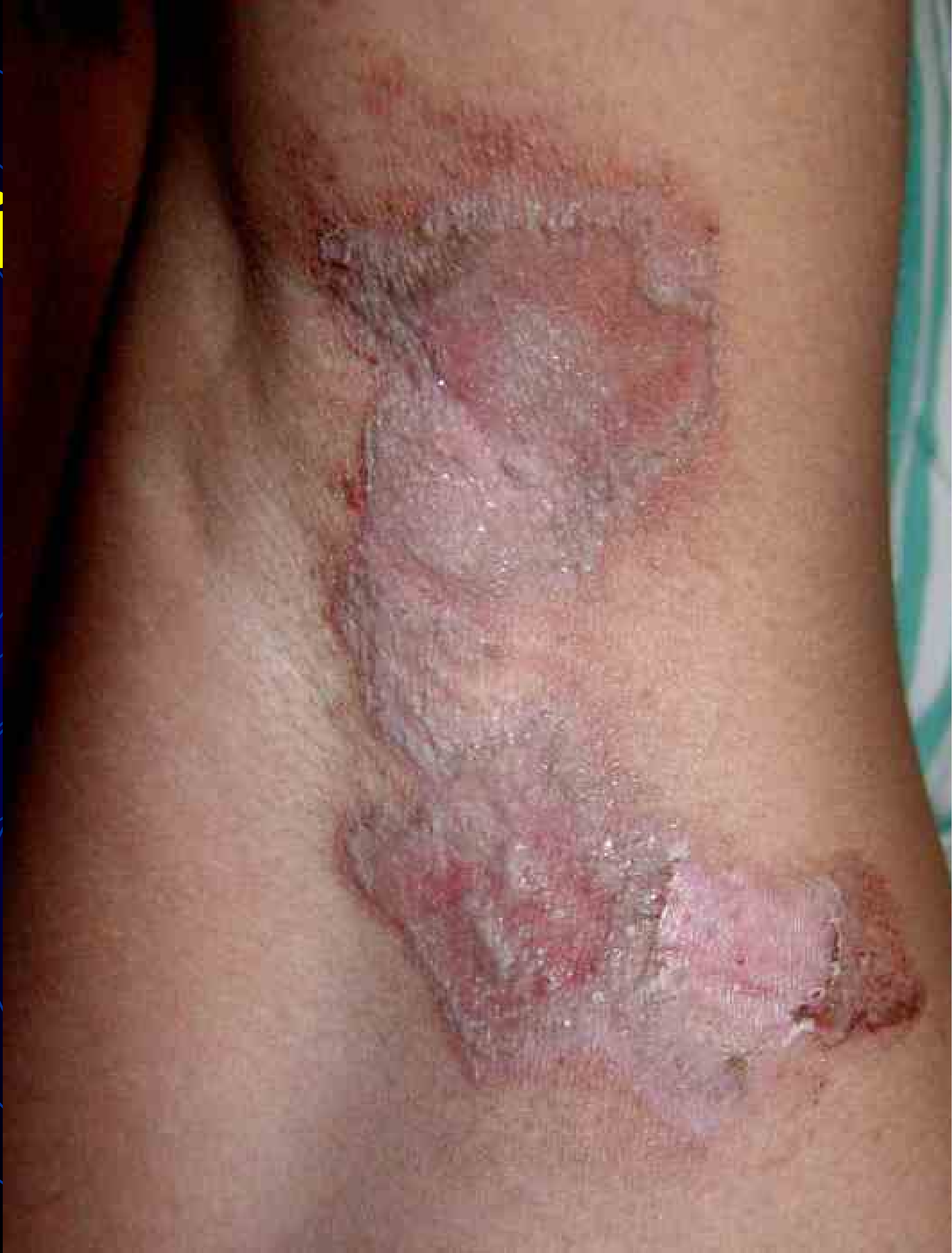
Diagnosis

- History. Site and morphology of the dermatitis. Patch testing in allergic contact dermatitis.

Treatment

- Detection and avoidance of the cause. The use of protective measures

Contact dermatiti



Atopic dermatitis

AD is an itchy, chronic, relapsing inflammatory skin condition, frequently associated with other atopic conditions in individual or other family members.

Clinical Picture AD starts at any age. Distribution of eruption varies with age.

Infantile phase (2 months- 2 years) The lesions frequently start on the face but may occur anywhere. The lesions consist of erythema and edematous papules. The papules are intensily itchy, and may become exudative and crusted.

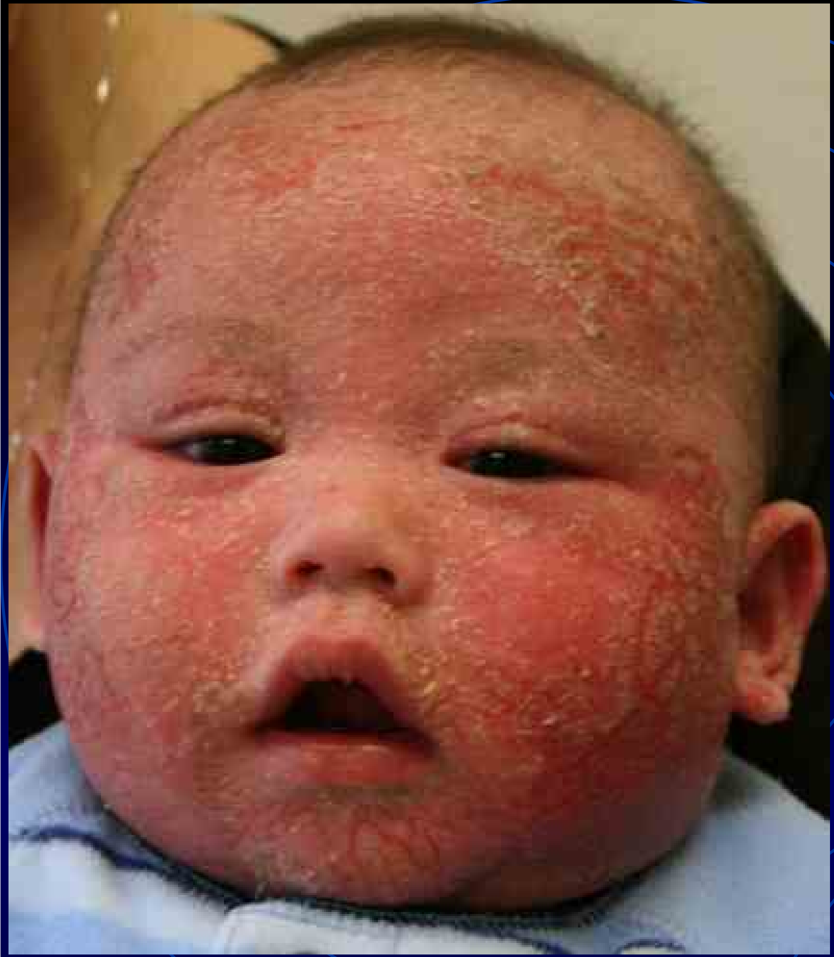
Childhood phase (2-12 years) The sites most commonly involved are the elbow and knee flexures, sides of the neck, wrists and ankles. The erythematous and edematous papules tend to be replaced by lichenification true eczematous lesions with vesiculation may occur. Itching is also intense.

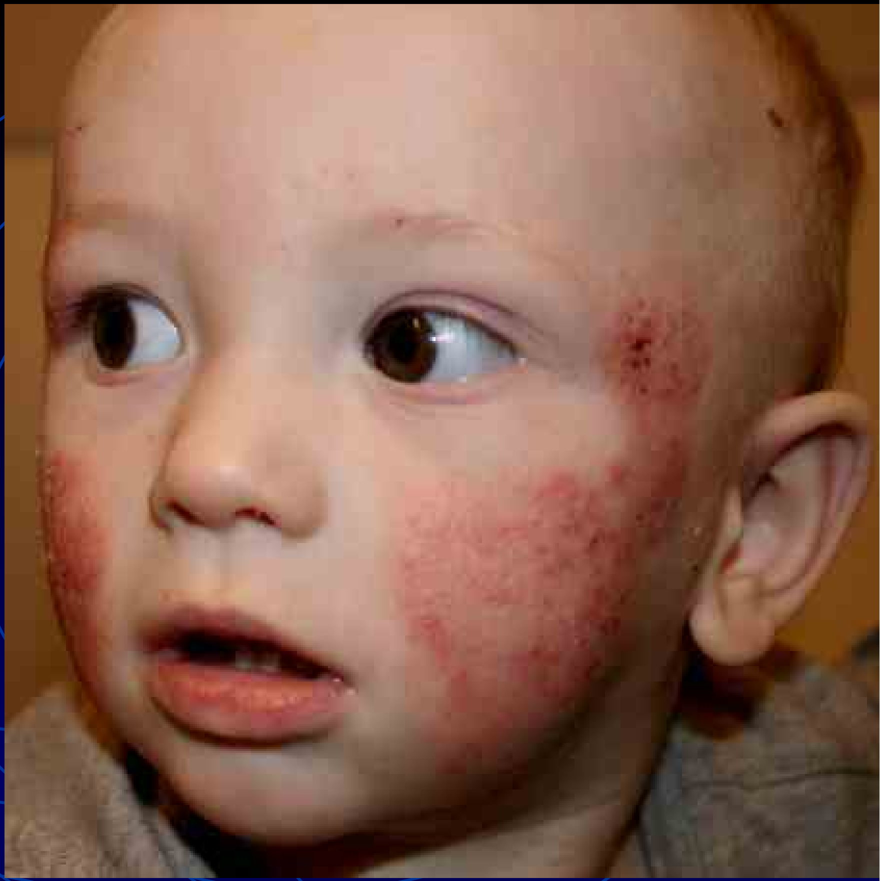
Adult phase (12 years and more) The picture is similar to that in later childhood, with lichenification, especially the flexures and hands.

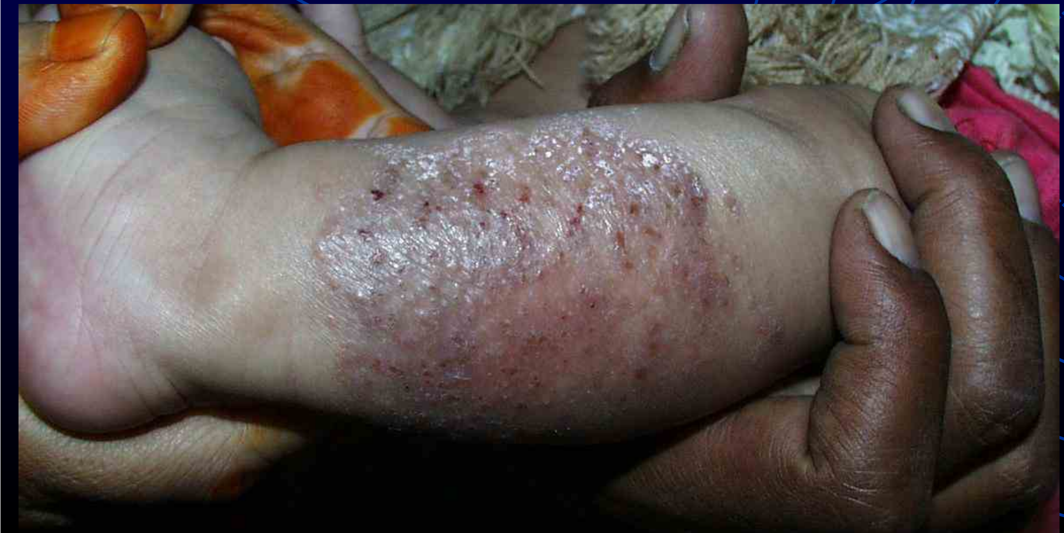
Treatment

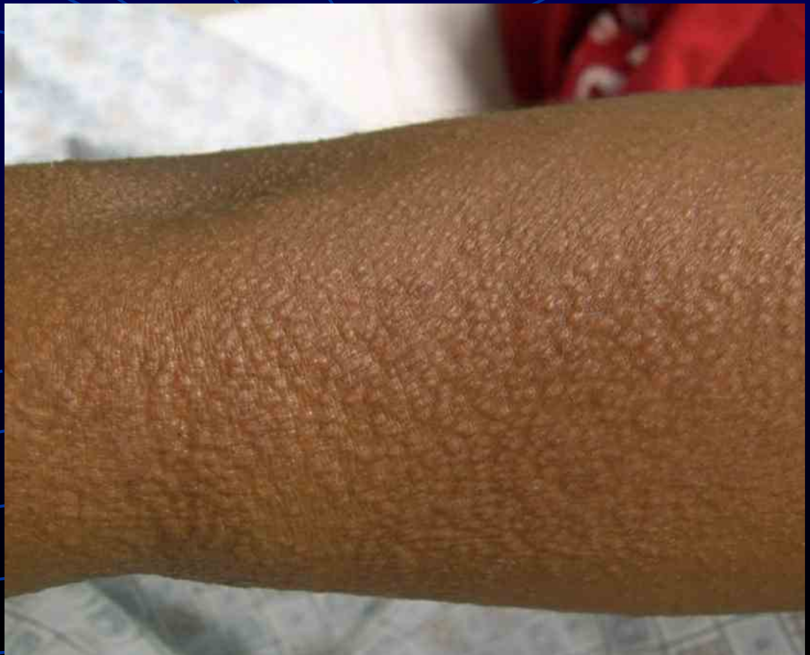
- Reduction of trigger factors as soap, detergents, wool, some

AD









Infective dermatitis

Infective eczema is eczema which is caused by microorganisms or their products, and which clears when the organisms are eradicated. Often it develops about a discharging abscess, sinus, or ulcer as an area of erythema with microvesicles.

Treatment

- Treatment of the infection in addition to treatment of the eczema.

Seborrhoeic dermatitis

- It is a chronic dermatitis, which has a red sharply marginated lesions covered with greasy scales and distributed in areas with a rich supply of sebaceous glands namely the scalp, face and upper trunk The yeast *Malassezia furfur* may have a role in the etiology of the disease.

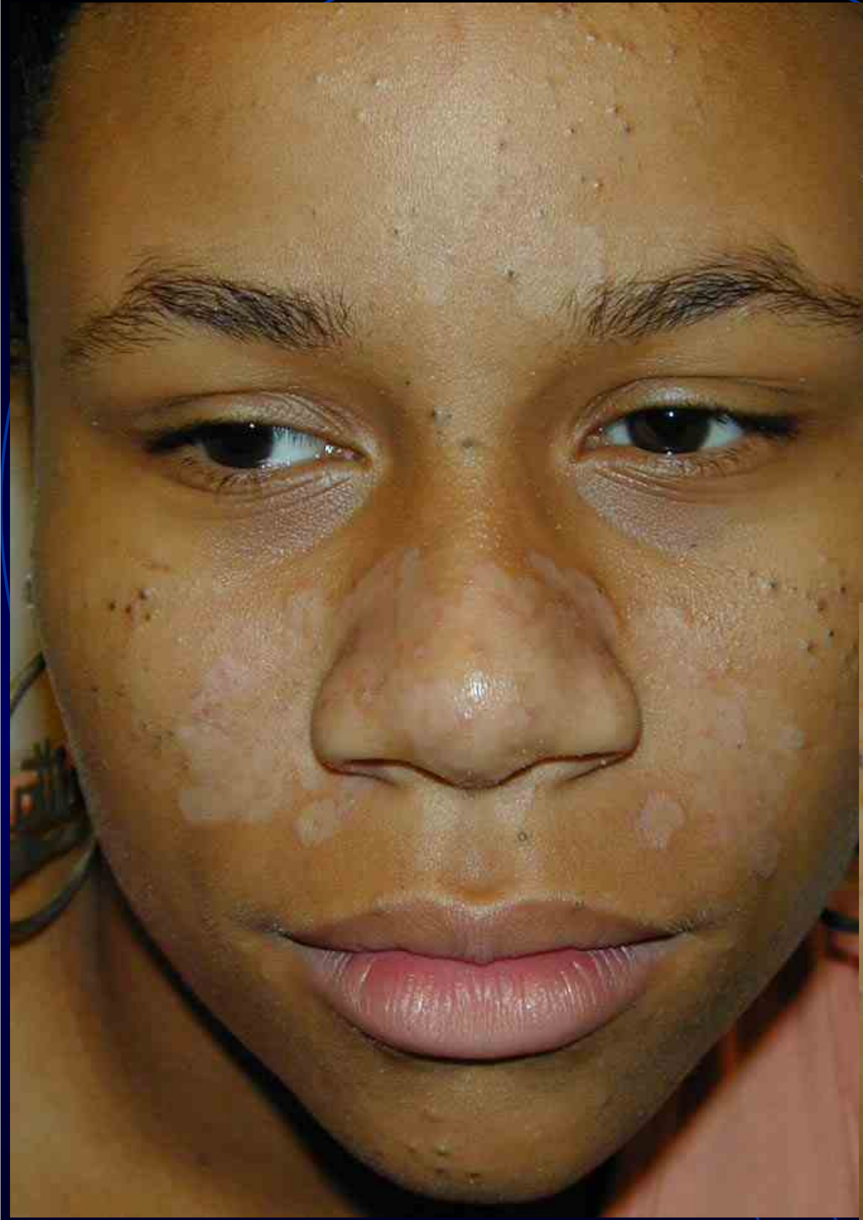
Clinical picture

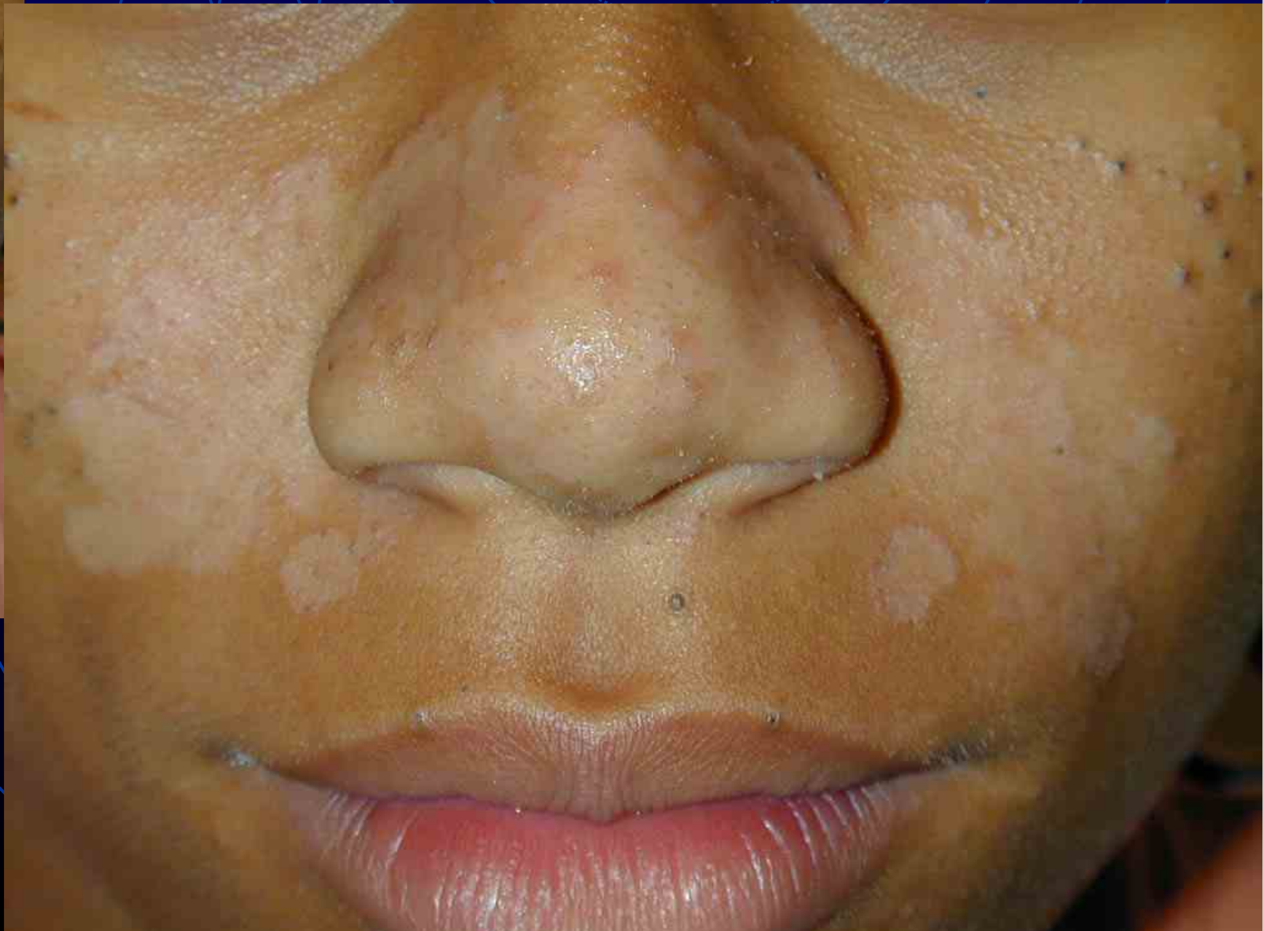
- **Dandruff** (visible desquamation from the scalp surface) appears to be the precursor of seborrhoeic dermatitis, and this may progress through redness, irritation and increasing scaling of the scalp to seborrhoeic dermatitis.
- **Seborrhoeic dermatitis** commonly originates in hairy skin, and involves the scalp, face, presternal and interscapular regions and the flexures. The lesions tend to be dull or yellowish red in color and covered with greasy scales. The lesions may be generalized, with erythema, scaling, oozing and pruritus and may progress to erythroderma.

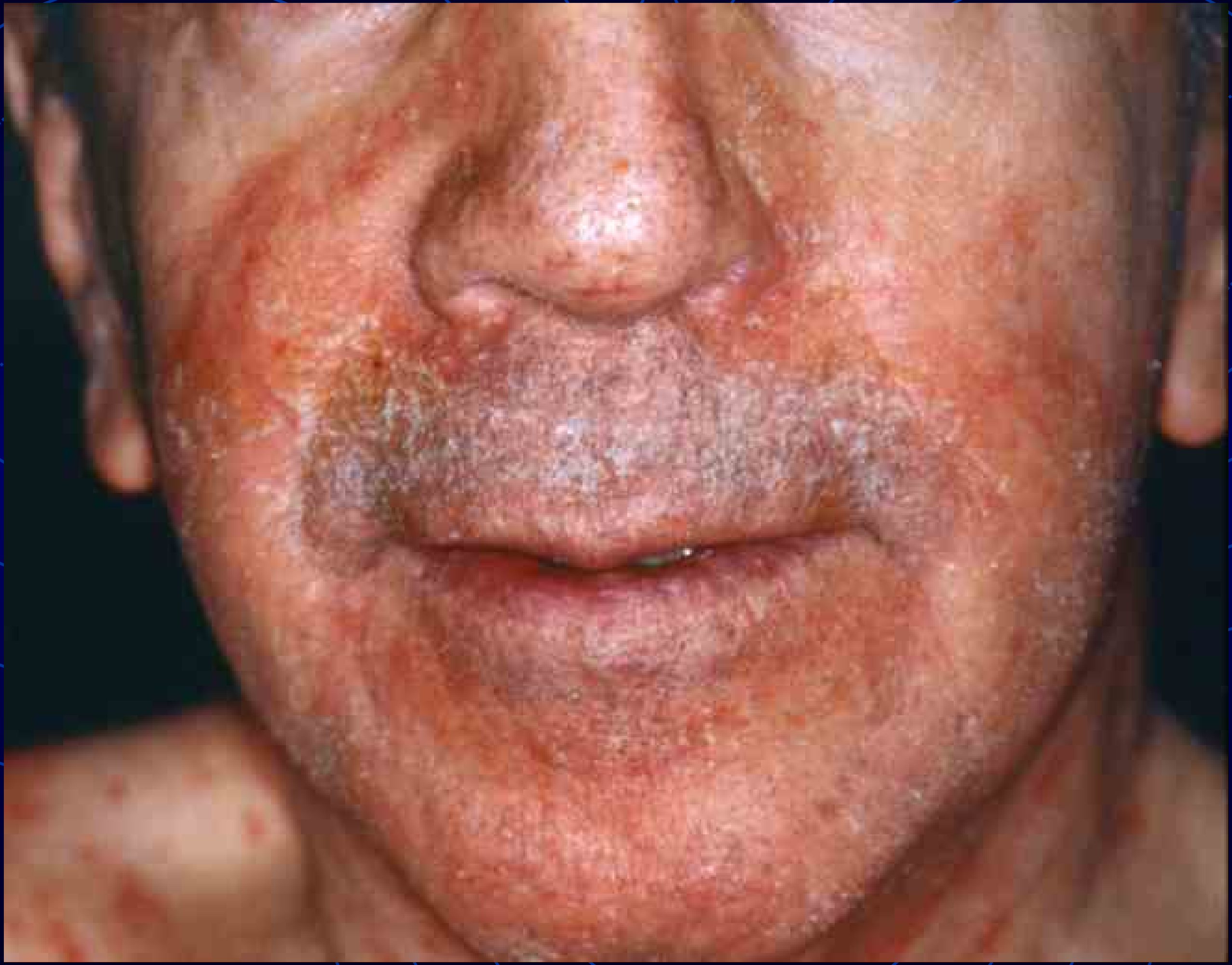
Treatment

- Dandruff is usually treated by medicated shampoos as

orrhoeic dermatitis







Discoid eczema (Nummular eczema)

- Discoid eczema is characterized by circular or oval plaques of eczema with a clearly demarcated edge.

Clinical picture

- The diagnostic lesion of discoid eczema is a coin shaped plaque of closely set, thin-walled vesicles on an erythematous base. Itching is usually severe. Middle aged men are most frequently affected. Discoid eczema usually begins on the lower legs, dorsa of hands, or extensor surfaces of the arm.

Pompholyx (Dyshidrotic eczema)

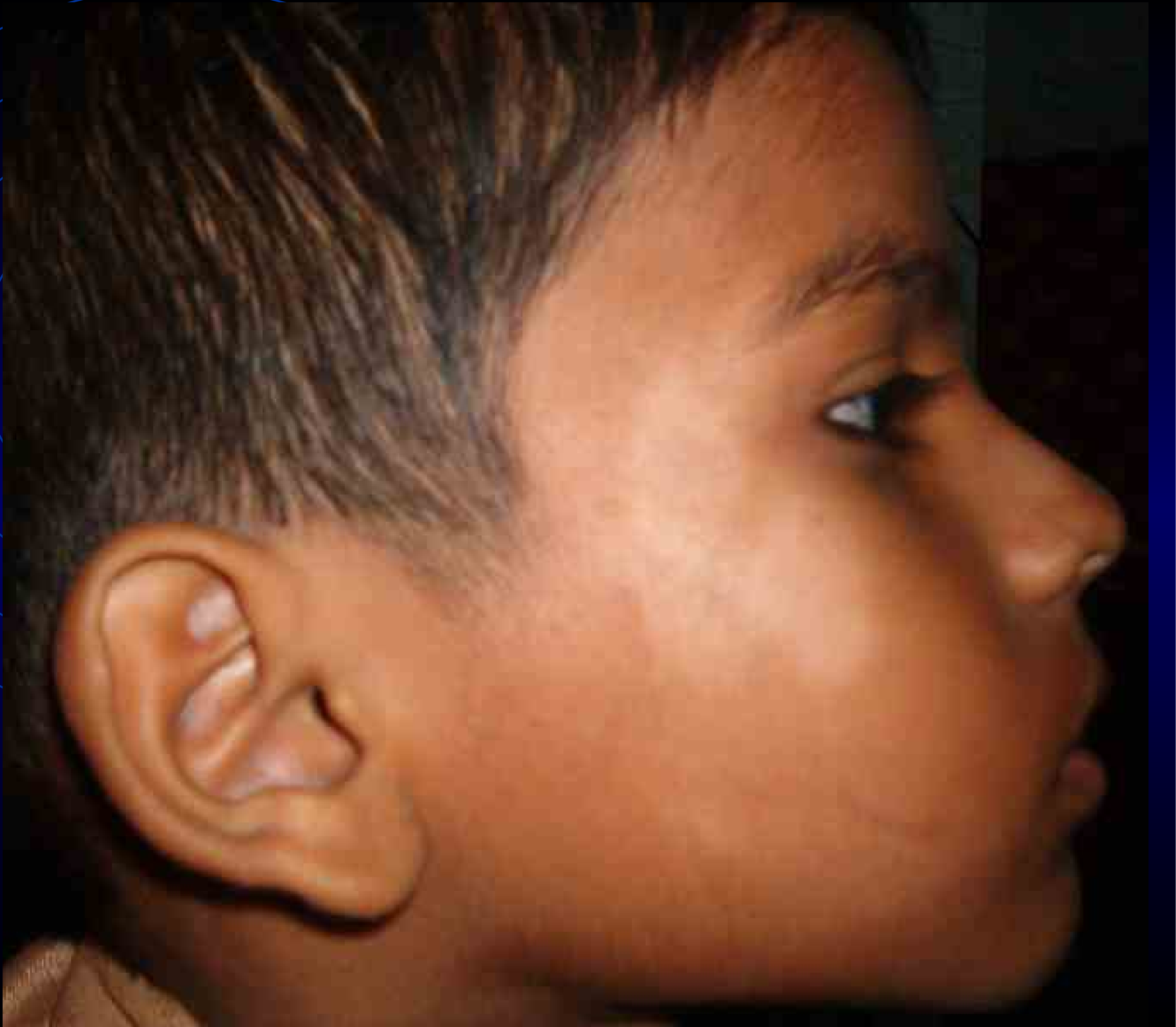
- Pompholyx is characterized by sudden onset of crops of clear vesicles, which appear deeply seated on the palms and soles. Vesicles may become confluent and present as large bullae. Itching may be severe. The attack subsides spontaneously, and resolution with desquamation occurs in 2-3 weeks.

Gravitational eczema (Venous or stasis eczema)

- Gravitational eczema is commonly secondary to venous hypertension, usually occurs in the lower leg and affects mainly middle-aged or elderly females. The eczema is usually accompanied by other manifestations of venous hypertension, including varicosity, edema, purpura, hemosiderosis and ulceration.

Pityriasis alba

- Pityriasis alba is a type of non specific dermatitis, of unknown origin. It is more common in children. It is characterized by erythematous scaly patches which subside to leave areas of hypopigmentation. The lesions are often confined to the face.



Treatment of eczema

General measures

- The cause should be removed or avoided.
- Antihistamines for itching.
- Antibiotics, local and systemic for secondary infection.
- Corticosteroids, local for the majority of cases and systemic for the severe, generalized or resistant cases.
- Wet dressings e.g. potassium permanganate 1/8000 are used for the acute cases.
- Creams are used for the subacute cases.

Urticaria

Urticaria is a transient eruption of erythematous or edematous swellings of the dermis and is usually associated with itching. The urticarial wheal results from increased capillary permeability, which allows proteins and fluids to extravasate.

Classification

- Urticaria may be acute with complete resolution within 6 weeks of onset or chronic lasting more than 6 weeks. It can also be classified into: immunologic, non-immunologic or idiopathic.

Etiologic factors

- Drugs e.g. penicillin, salicylates, morphine, nonsteroidal anti-inflammatory drugs and codeine.
- Foods e.g. seafood, strawberries, chocolate, Food additives.
- Insect bites and stings.
- Physical agents e.g. heat, cold and physical injury.
- Inhalants e.g. nasal sprays, pollens and animal danders.
- Infections e.g. focus of infection in the sinuses, teeth or tonsils.
- Parasitic infestations e.g. ascaris and ankylostoma.
- Emotional stress. It is important in cholinergic urticaria.

Clinical picture

Itchy erythematous macules develop into wheals. They occur anywhere on the body, last a few hours and resolve within 24 hours. Mucosal swellings occur inside the oral cavity, tongue, pharynx and larynx.

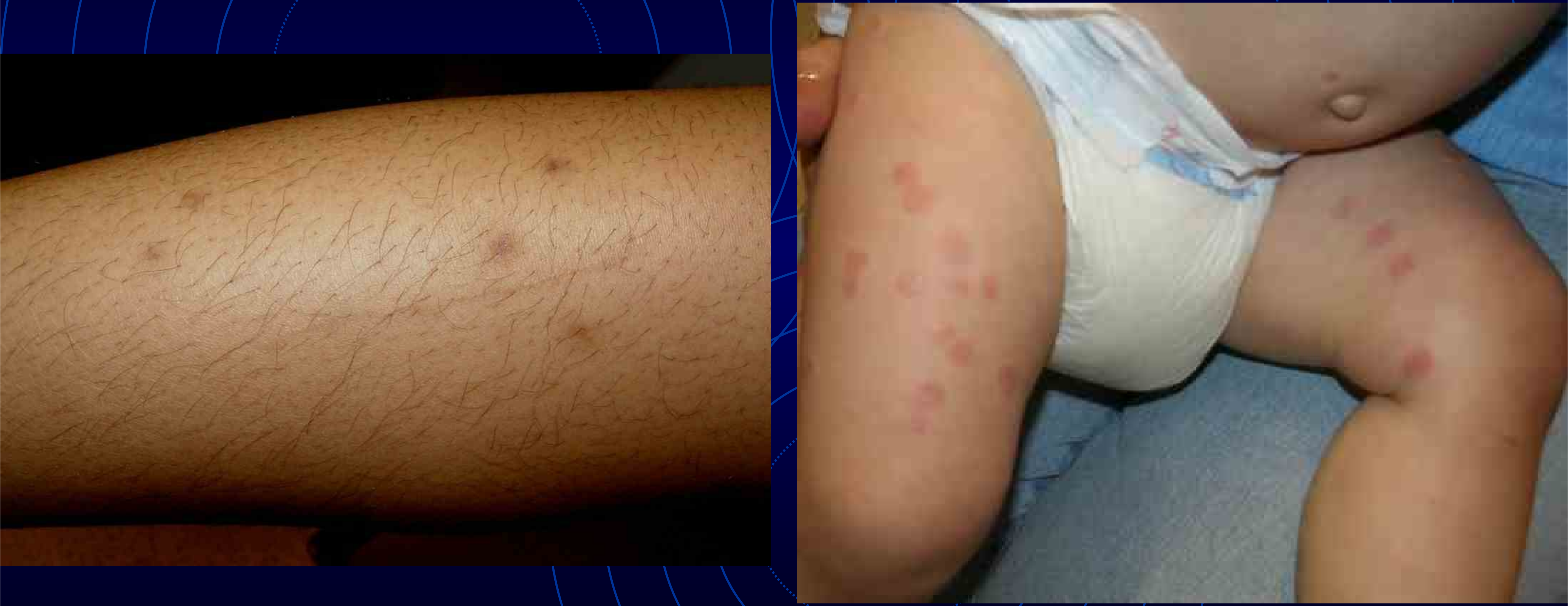
Special varieties of urticaria

- **Papular urticaria.**
- **Physical urticaria** e.g. dermographism, cholinergic urticaria, cold urticaria.
- **Angioedema (angioneurotic edema)** is a type of acute urticaria affects the most distensible tissues e.g. eyelids, lips, lobes of the ears and external genitals with circumscribed edema. The swelling affects the deeper parts of the skin or the subcutaneous tissue with the overlying skin unaltered. It may also affect the mucous membranes. Affection of the larynx is a life threatening condition.

Treatment

- Detection and avoidance of the cause.
- Antihistamines.

Papular urticaria



Papular urticaria

It is a characteristic reaction to insect bites as mosquitoes or fleas.

Clinical picture

- The lesions develop mainly on the upper and lower extremities particularly the forearms and legs. Initially, an extremely itchy urticarial wheal develops at the site of the bite, and this is succeeded by a firm pruritic papule, which may be surmounted by a tiny vesicle. Itching is an almost constant symptom and secondary infection is a common complication. Usually subsides spontaneously.

Treatment

- Control of insects. Antihistamines. Topical antipruritics e.g. calamine.

Prurigo of Hebra It is a pruritic papular eruption believed to be a continuation of papular urticaria.

Clinical Picture

- It is a disease of children and affects mainly the extensor surfaces of the extremities but in long standing cases, other areas may be affected. Lichenification and Lymphadenopathy

Erythema multiforme

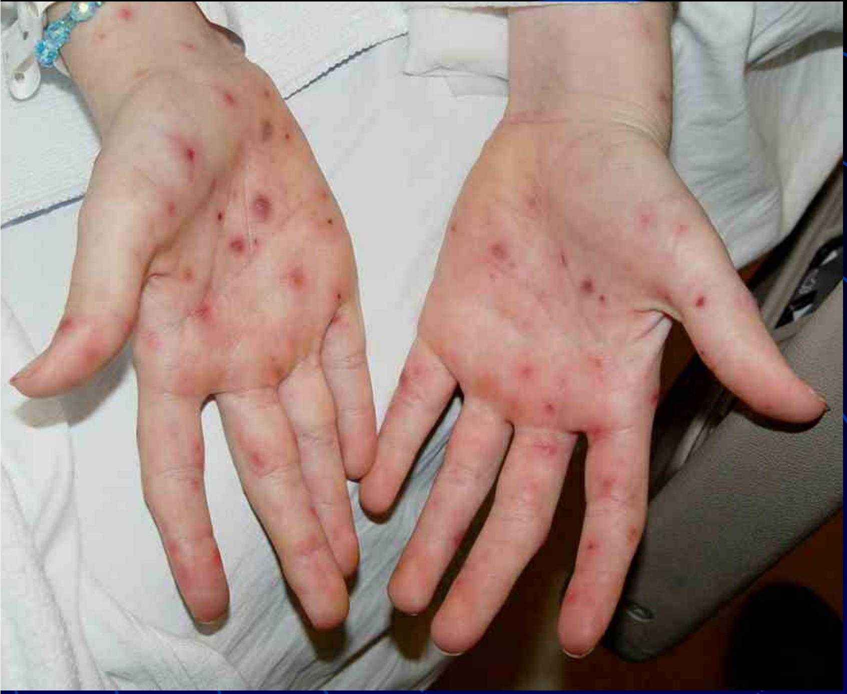
EM is an acute self-limited eruption of the skin and m.m. by the distinctive target or iris lesion

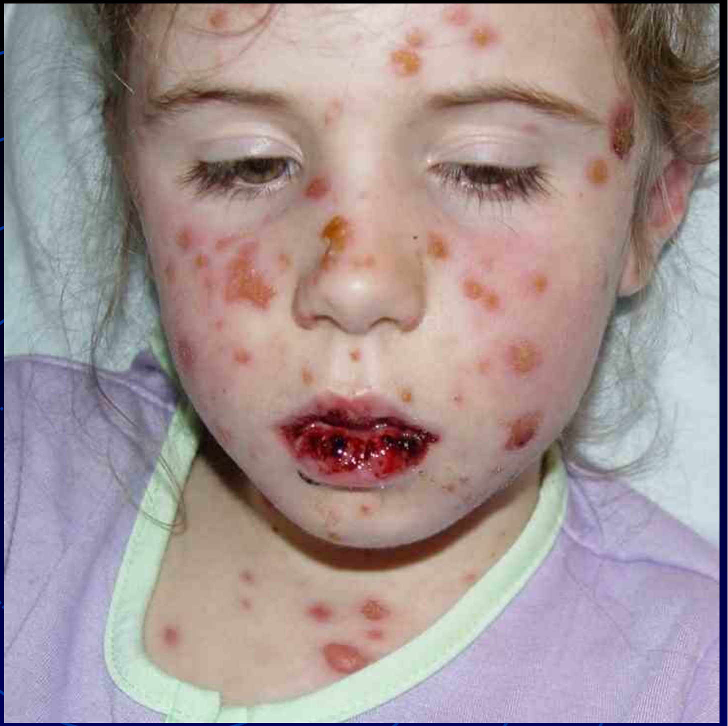
Etiology

- The precise etiology is unknown. The trigger factors are:
- Viral infections Mycoplasma pneumoniae. X-ray therapy.
- Drugs. Lupus erythematosus. Carcinoma, lymphoma.

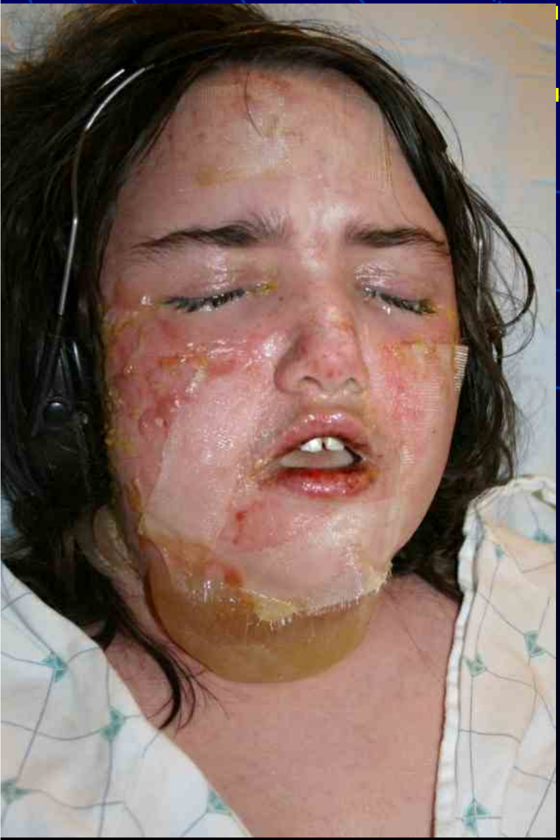
Clinical picture

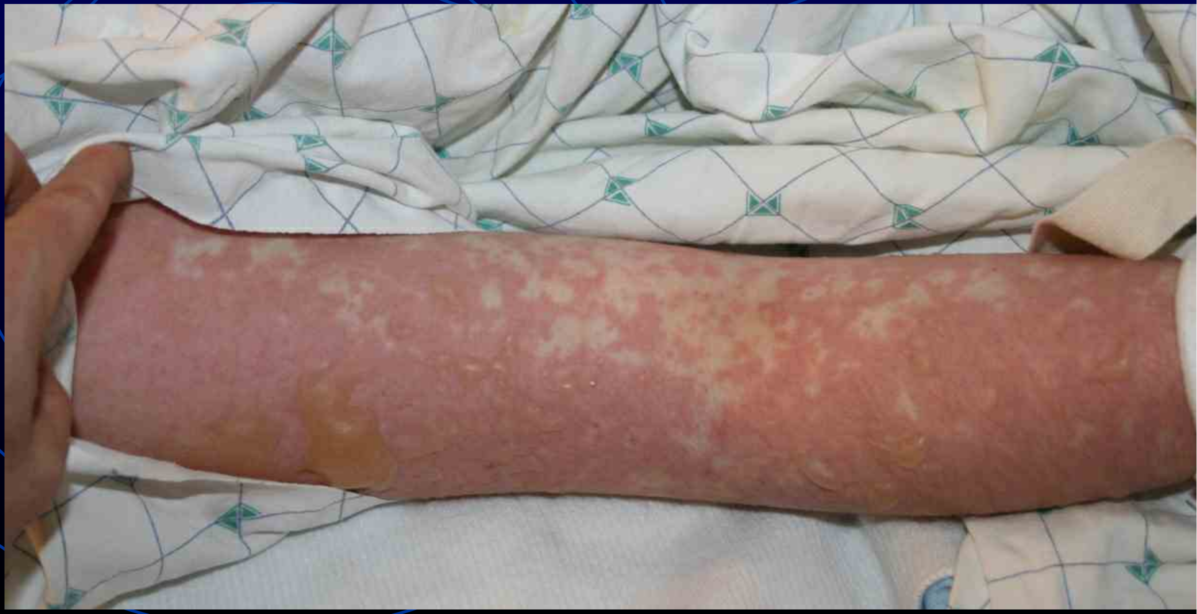
- **Erythema multiforme minor.** This accounts for about 80% of the cases. It occurs seasonally in the spring and fall. The lesions are dull red maculopapules in addition to the characteristic target or iris lesions. A typical target lesion has 3 zones: a central area of dusky erythema or purpura, middle paler zone of edema and an outer ring of erythema. Classically, the backs of the hands, palms, wrists, feet and extensor aspects of the elbows and knees are affected, less commonly the face. There may be erosions or bullae on the mucous membranes
- **Erythema multiforme major (Stevens-Johnson syndrome).** It is the severe form of the disease and frequently accompanied by a febrile prodrome. Mucous membranes show extensive bullae formation and erosions. The skin lesions may be in the form of maculopapules, bullae or pustular lesions.
- **Treatment**





rythema multiforme





Drug eruptions

Drug eruptions are probably the most frequent of all manifestations of drug sensitivity. They may arise as a result of immunological allergy directed against the drug itself, a reactive metabolite or some contaminant of the drug or more commonly, by non-immunological mechanisms.

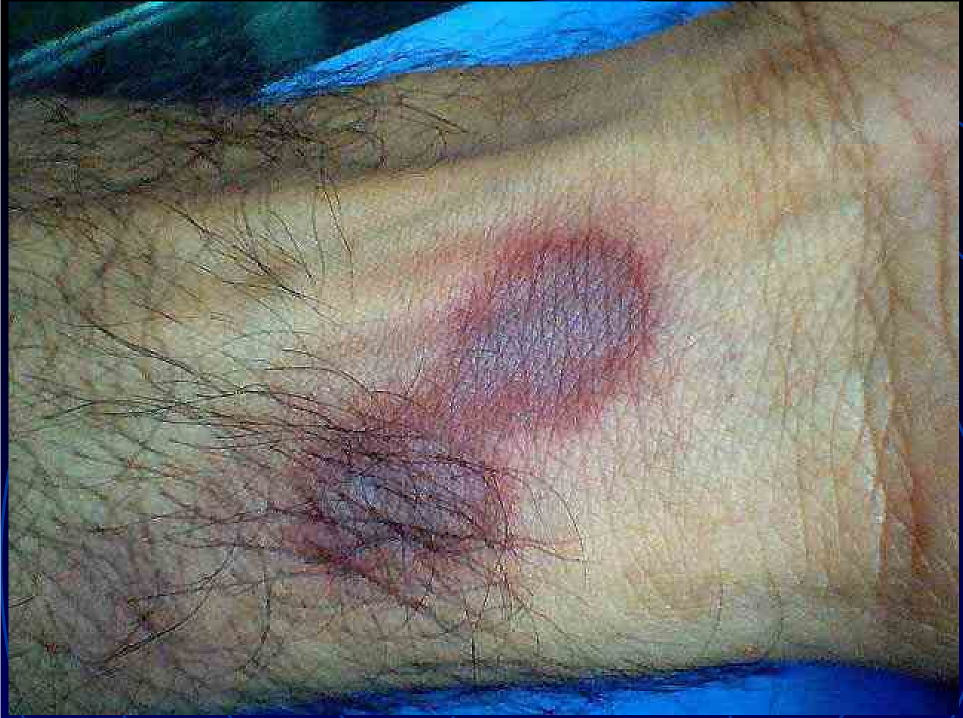
Fixed drug eruption

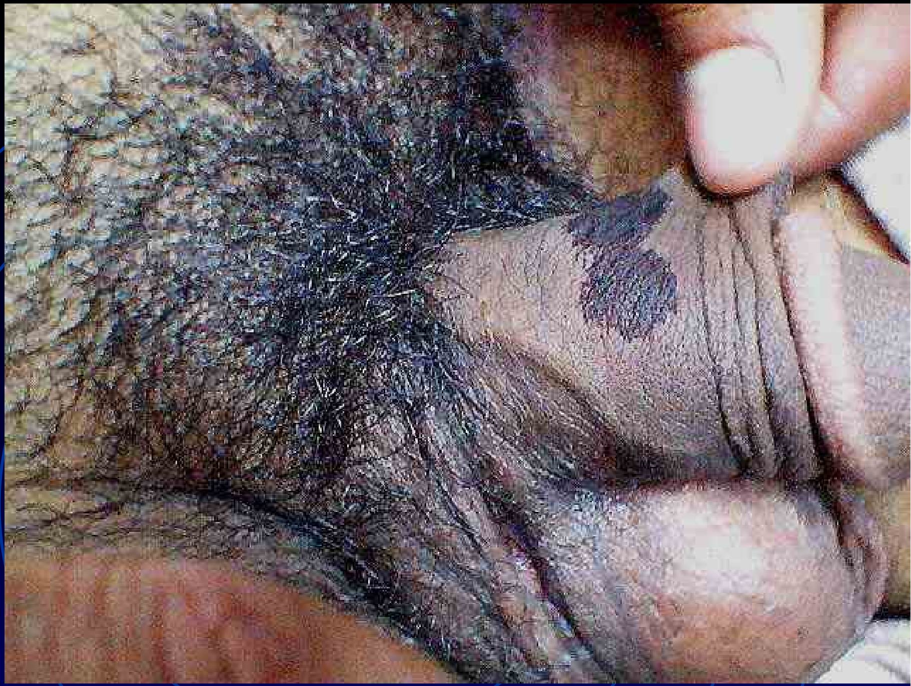
Fixed drug eruptions characteristically recur in the same site or sites each time the drug is administered. The drugs commonly cause this eruption are: sulphonamides, tetracyclines,

Clinical picture

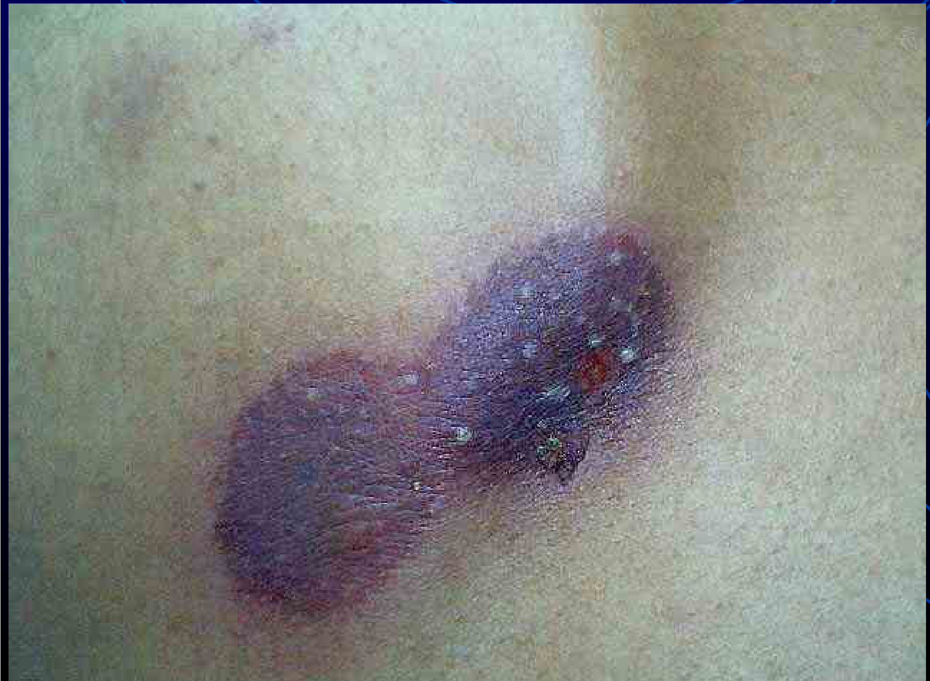
- The lesion usually presents as sharply marginated, round or oval itchy plaques of erythema and edema becoming dusky violaceus or brown, and sometimes vesicular or bullous. Lesions are commoner on the limbs, but about half the cases occur on the oral and genital mucosa. Characteristically prolonged post-inflammatory hyperpigmentation results.

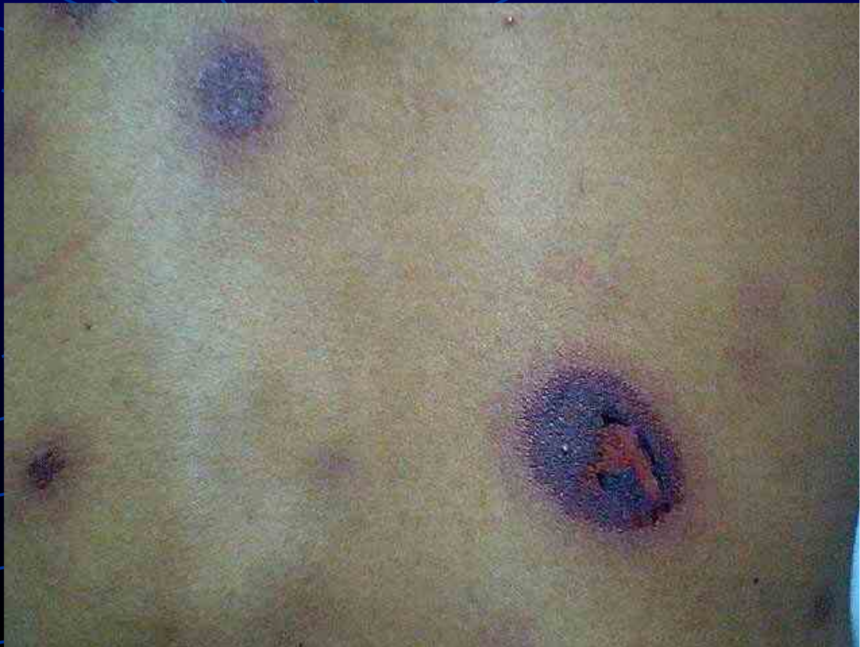
Treatment





Fixed drug eruption





Erythematosquamous diseases

Abdelwassa Almekhlafi,MD

Psoriasis

Psoriasis is a common, chronic, recurrent inflammatory disease of the skin characterized by round, circumscribed, erythematous, scaly papules and plaques of various sizes, covered by silvery white scales.

Etiology

The etiology is unknown; the suggested causes are:

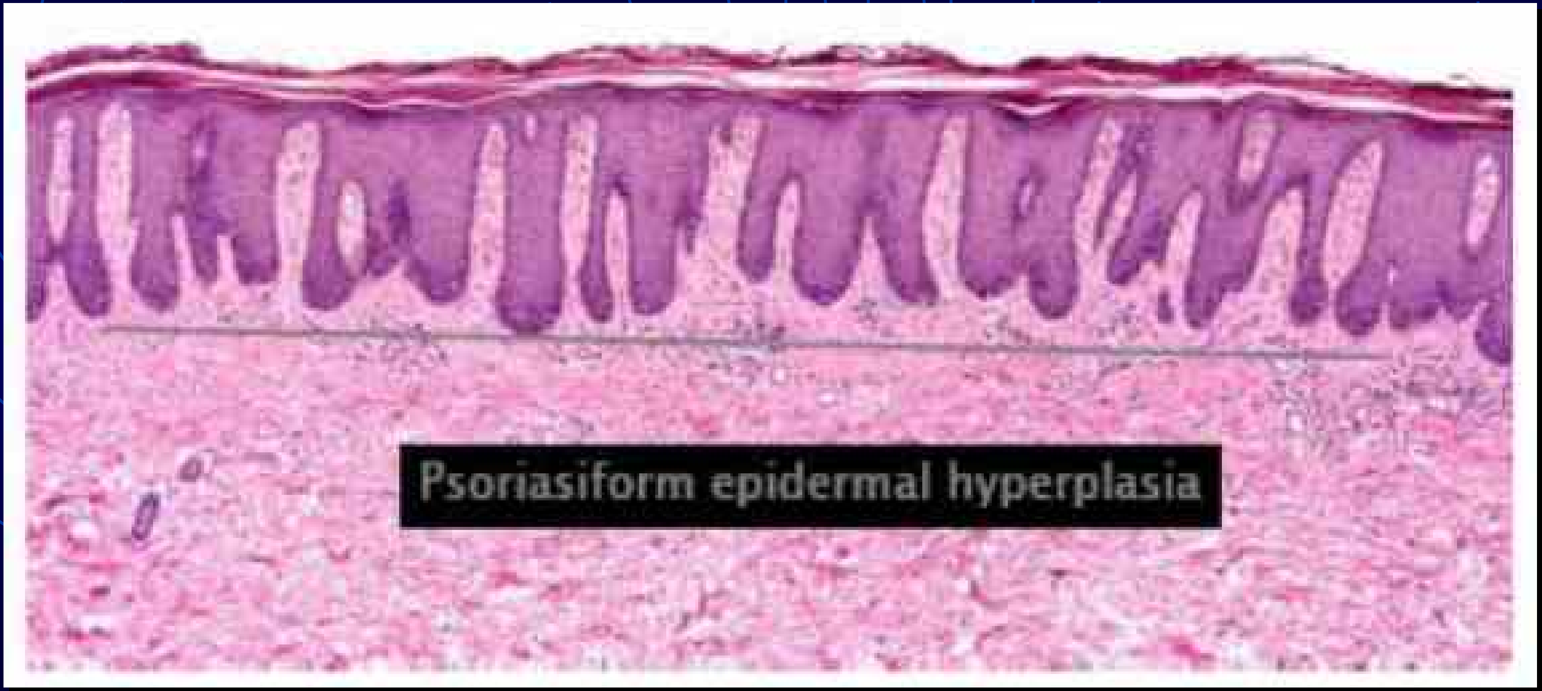
- Genetic predisposition; family history is common.
- Epidermal hyper-proliferation.
- Vascular proliferation.
- Immunological and inflammatory abnormalities.

Provocating factors :

- **Trauma :** All types of trauma can lead to the development of plaque psoriasis. The development of psoriatic lesions at a site of injury is known as the **Koebner phenomenon**
- Infection:An acute eruption of guttate psoriasis may be provoked by streptococcal pharyngitis.
- Drugs. Lithium ,withdrawal of systemic corticosteroids, beta-blockers, antimalarials, and NSAIDs may cause flare of the disease
- Sunlight: generally considered to be beneficial for most of the patients, strong sunlight may worsen the disease.
- Stress - Many patients report an increase in the psoriasis severity with psychological stress.
- **Alcohol - Alcohol is considered a risk factor for psoriasis.**
- **Endocrine – the disease state may fluctuate with hormonal changes. Psoriasis may begin during puberty. Pregnancy may improve the disease. while a flare may occur during post-partum period.**

pathology

- The pathology of psoriasis reflects the underlying immune-mediated inflammation and cellular hyperproliferation.
- Hyperkeratosis with parakeratosis (presence of nucleated keratinocytes in the stratum corneum due lack of maturation of cells since rapid transit time do not permit normal maturation of cells).
- Reduced or absent granular layer.
- Acanthosis with elongation of rete ridges and a corresponding upward elongation of dermal papillae.
- Infiltrate: Mononuclear in dermis and polymorphs in the upper epidermis forming collections called 'microabscess of Munro'.
- Upper dermal vasculature shows dilatation and tortuosity.


Psoriasiform epidermal hyperplasia

Classification:

1-non pustular ps 2- pustular ps

Clinical picture (non pustular ps)

- Psoriasis is an asymptomatic disease but itching may be present in acute spreading cases and the flexural type.
- The characteristic lesions are erythematous papules and plaques covered with dry, silvery-white scales. The eruption is usually **bilateral** and **symmetrical** and may vary from a solitary lesion to more than 100.
- Any site can be affected but the commonest are; **extensor surfaces of the limbs, elbows, knees, sacral region, scalp and nails.**
- The course of the disease is chronic and unpredictable.
- The lesions are covered with a silvery white, mica-like, loosely adherent scales which, on removal may reveal punctate bleeding points **(Auspitz sign)**

Clinical types

- **Psoriasis vulgaris.** Classical type, previously described.
- **Guttate psoriasis.** -Guttate psoriasis generally follows a **streptococcal** infection and presents with an acute symmetrical eruption

  -Lesions are the size of water drops.
- **Discoid (Nummular) psoriasis.** Coin-shaped lesions.
- **Annular psoriasis.** Ring shaped lesions.
- **Psoriasis of the scalp.** Multiple plaques or diffuse involvement with no loss of hair.
- **Psoriasis of the nails.** Pitting, loss of translucency, onycholysis, subungual hyperkeratosis.
- **Flexural psoriasis.** Affects the flexures as the groins and axillae. Due to friction and moisture scaling is reduced or absent and itching is common.
- **Pustular psoriasis.** May be localized or generalized. The pustules are sterile.
- **Erythrodermic psoriasis.** Erythema and scaling involving almost all the skin surface.
- **Arthropathic psoriasis.** Psoriasis associated with arthropathy. It may be in the form of distal arthritis (distal

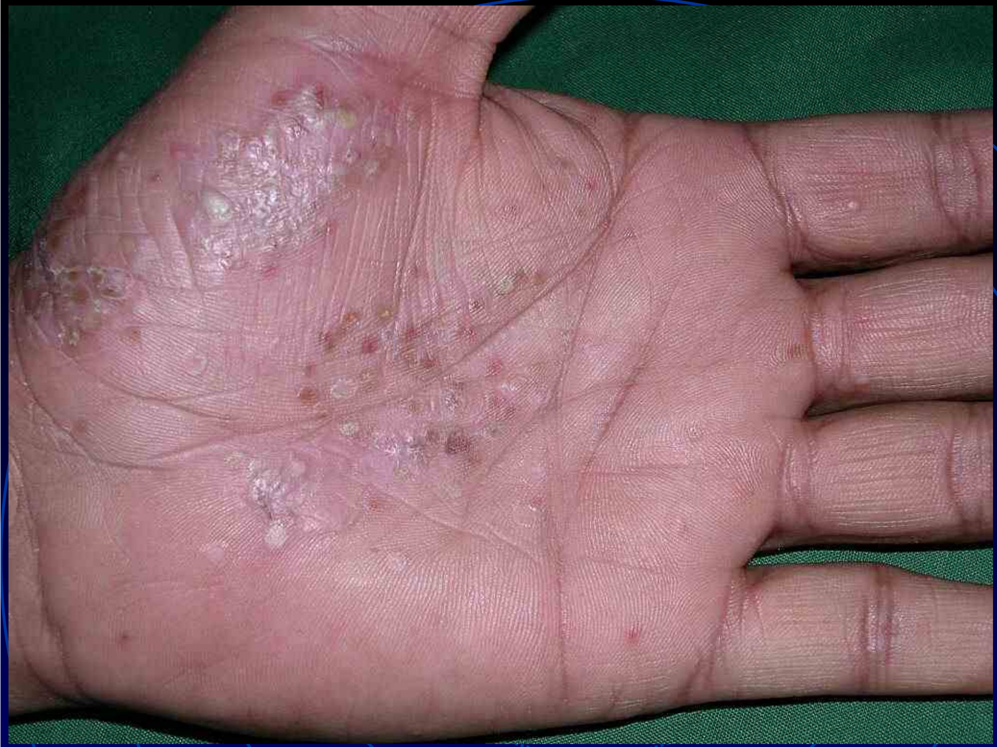
Variations by site:

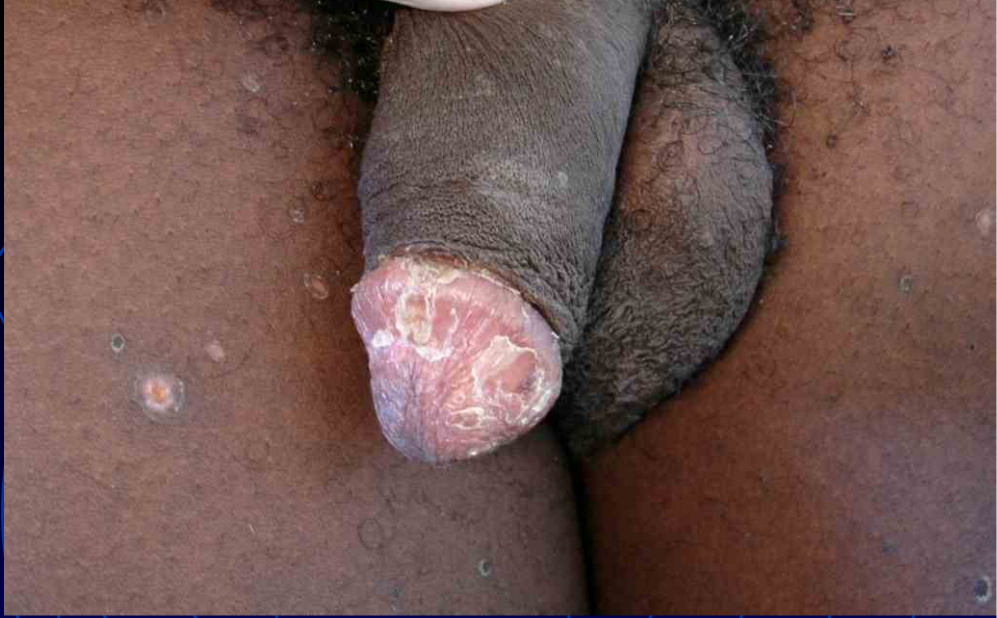
- **Scalp:** Diffusely involved. Thick scaling, no hair loss.
- **Penis:** Well-circumscribed reddish plaque without scales.
- **Hands and feet:** Diffuse hyperkeratosis, Thick scales, fissures.
- **Sacral regions:** thick fissures plaques, scaling may be absent.
- **Nails:** involved in 25 to 50% of cases.

-Nail pitting. -Discoloration. -Subungual hyperkeratosis.

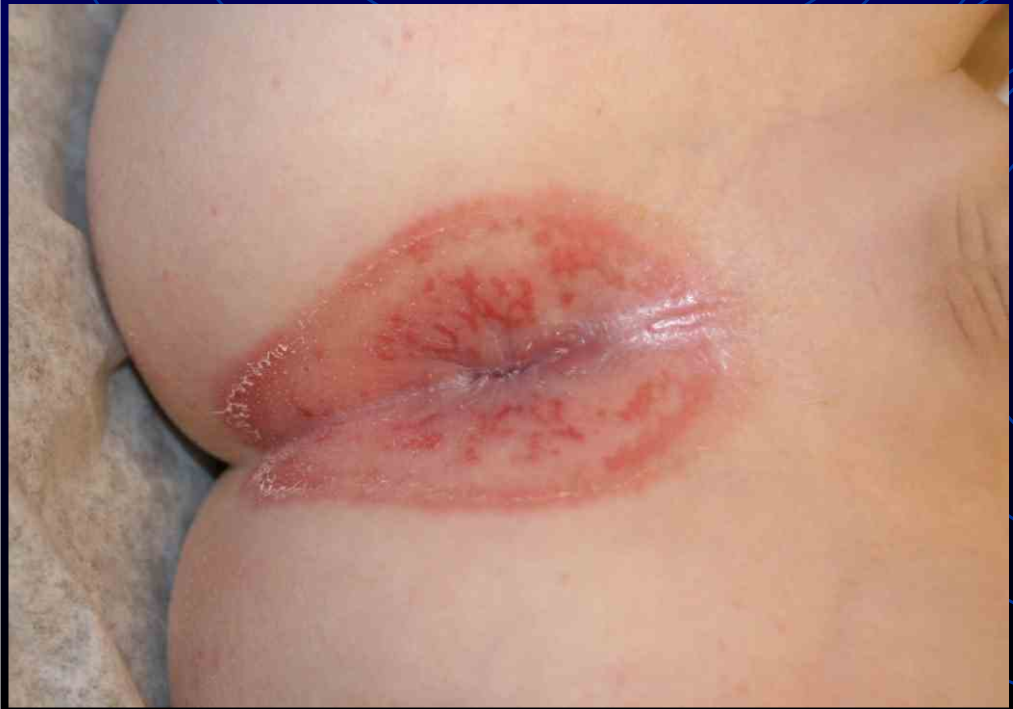
-Onycholysis (separation of nail-plate from the nail bed) giving rise to oil-drop sign.

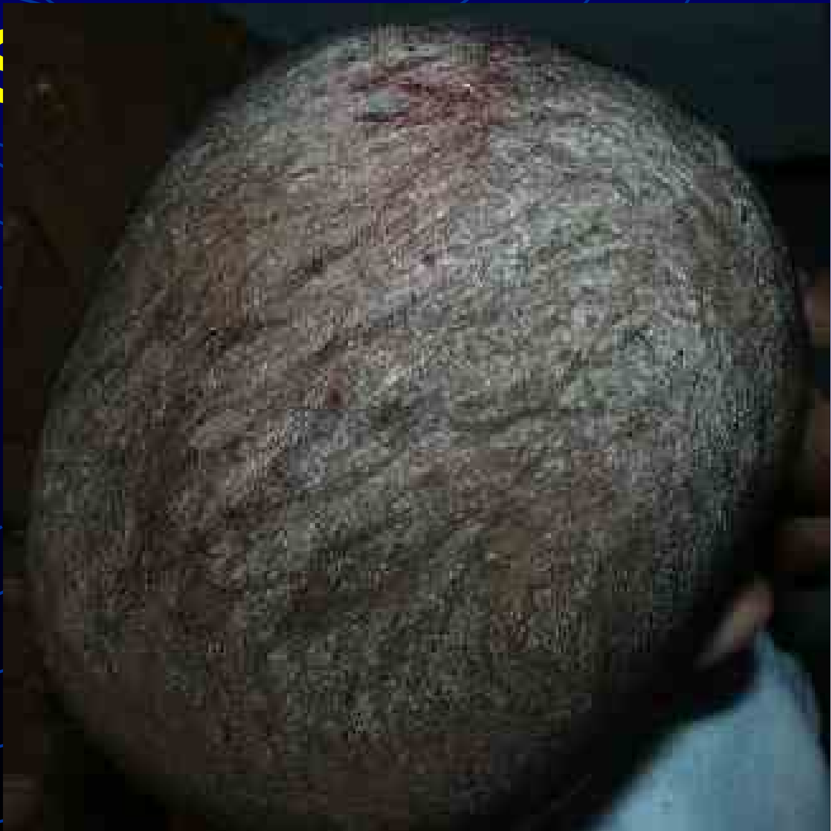
-Thickened friable nail plate.



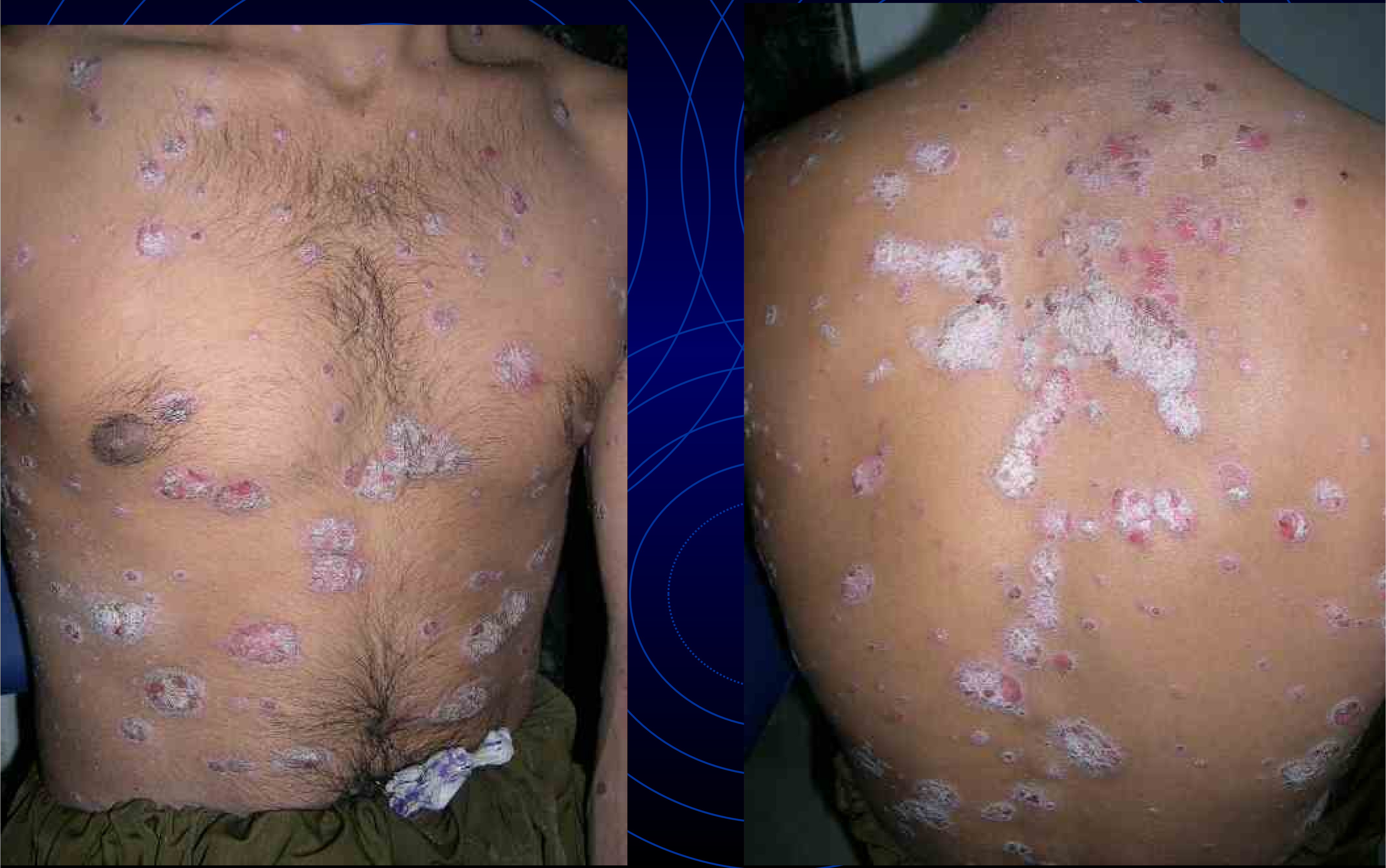


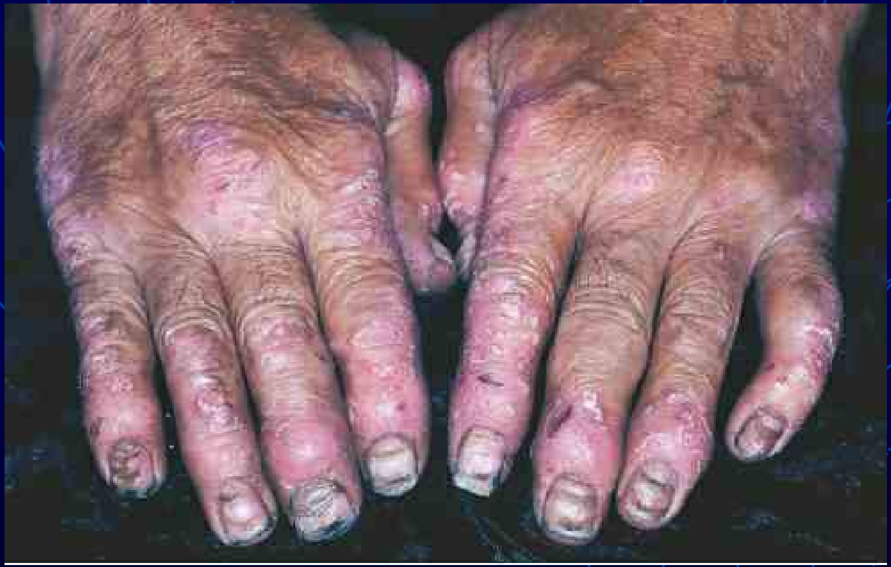
Psoriasis

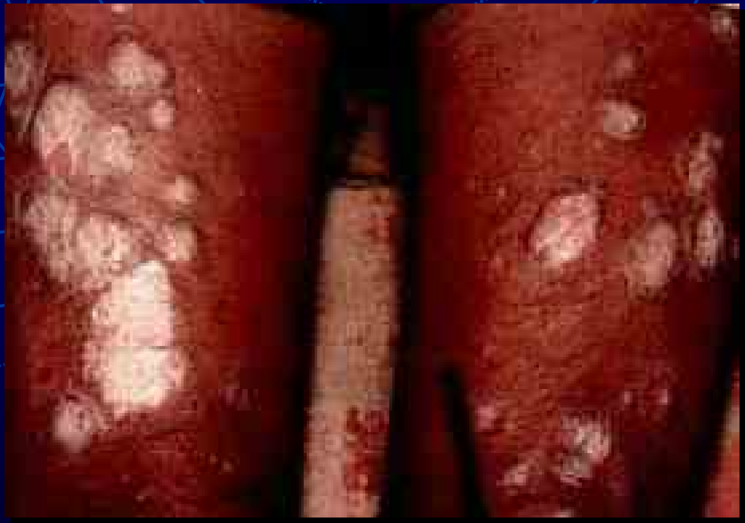




Psoriasis







DIFFERENTIAL DIAGNOSIS

Diagnosis of psoriasis is usually based on typical clinical features alone. However, in certain cases, depending on the type and site of lesions, the following diseases may need to be considered in the differential diagnosis.

-Seborrhoeic dermatitis
-Pityriasis rubra pilaris
-Leprosy
-Candidiasis
-Discoid lupus erythematosus
-Hyperkeratotic eczema of hands and feet
-Pityriasis rosea
-Lichen simplex
-Secondary syphilis
-Lichen planus
- Tinea corporis and cruris

Treatment

A- Topical therapy

- Corticosteroids. It is the most frequent therapy, used with occlusion is more effective. Intralesional injection of steroids is very useful in small lesions.
- Tars. Crude coal tar 2-5%.
- Salicylic acid. 3-5%.
- Vitamin D3 analogue (Calcipotriol).

B- PhOTHERAPY: PHOTPhototherapy is used in the cases of extensive, widespread disease resistant to topical treatment .

- **UVB - Ultraviolet B** (UVB) irradiation utilizes ultraviolet radiation with wavelengths 290-320 nm (visible light range is 400-700 nm). It is used alone or combined with one or more topical treatments. The Goeckerman regimen uses coal tar followed by UVB exposure and the Ingram method is based on anthralin application following a tar bath and UVB treatment. UVB more commonly is now combined with topical corticosteroids, calcipotriene, tazarotene, or simply bland emollients. UVB phototherapy is extremely effective for treating moderate-to-severe plaque psoriasis. Narrow-band UVB (UVB of 311 nm wave length) is now increasingly used for its effectiveness and low potential for photodamage.

- **PUVA photochemotherapy :** In this therapy (also known as PUVA), a photosensitizing drug psoralen (8-methoxypsoralens) is given orally, followed by ultraviolet A (UVA) irradiation to treat patients with more extensive disease. UVA irradiation utilizes light with wavelengths 320-400 nm. PUVA, decreases cellular proliferation by interfering with DNA synthesis, and also induces a localized immunosuppression by its action on T lymphocytes... Therapy usually is given 2-3 times per week on an outpatient basis, with maintenance treatments every 2-4 weeks until remission. Adverse effects of PUVA therapy include **nausea, pruritus, and burning.** Long-term complications include **increased risks of photo damage and skin cancer.** PUVA has been combined with oral retinoid derivatives to decrease the cumulative dose of UVA radiation to the skin.
- Eximer laser (308 nm)

c- Systemic therapy: Systemic treatments are used when both topical treatments and phototherapy have failed. Also used for patients with very active psoriatic arthritis, erythrodermic psoriasis and generalized pustular psoriasis.

- **Antimetabolites:Methotrexate** (a folic acid antagonist). is the most popular agent used. A weekly oral or intramuscular dose of 15 to 25 mg is used. Main side effects are nausea, vomiting, hepatic dysfunction
- **Retinoids: Acitretin** (a synthetic vitamin A derivative.) s used in a dose of 10 to 20 mg/day. Main side effects are : teratogenicity (the drug should not be used in women of child-bearing potential) , hypertriglyceridemia, xerosis of skin, chelitis and alopecia
- **Cyclosporine A** (immunosuppressive drug). The usual dosage is 3 to

COMPLICATIONS

-Complications are relatively uncommon.
-Many of the complications (pustular psoriasis, erythroderma) are commonly due to inappropriate and aggressive therapy.

- Psoriatic arthritis.
- Pustular psoriasis.
- Erythroderma and its metabolic complications.
- Infection, particularly Staph. infections of the patches.
- Eczematization due to topical agents.
- Psychological consequences : depression, anxiety, lack of self-esteem.
- Potential complications of systemic therapy should not be overlooked.

PROGNOSIS

- The course of plaque psoriasis is **unpredictable**. The duration of active disease, the time or the frequency of relapses, or the duration of a remission are unpredictable.. The disease rarely is life threatening, but often is intractable to treatment with relapses occurring in the majority of patients.
- Both early onset and family history of disease are considered poor prognostic indicators.

Lichen planus

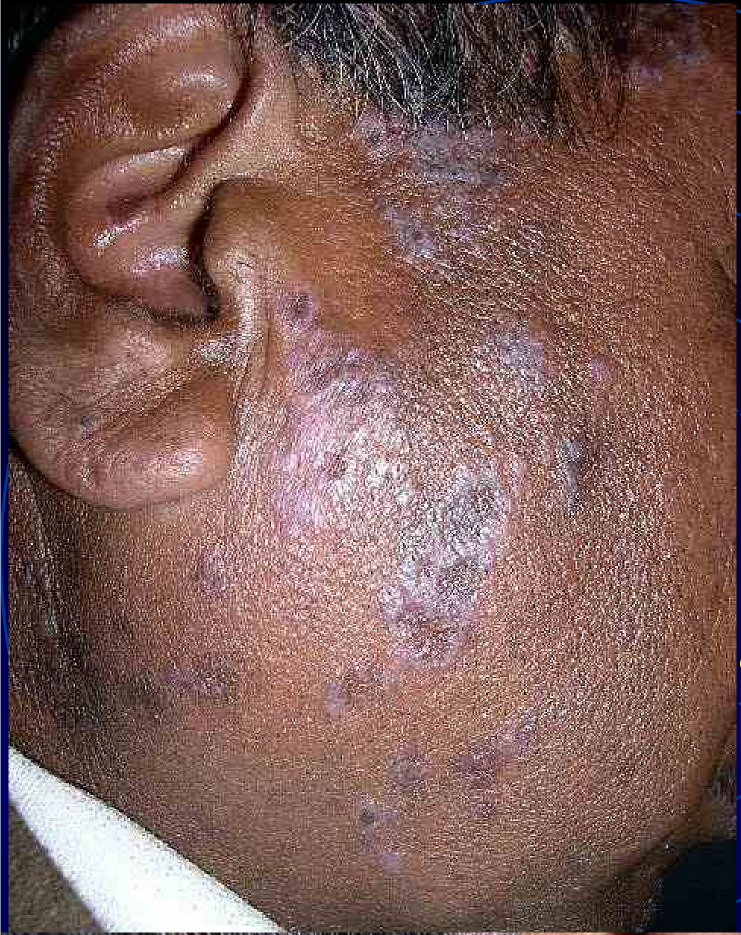
Lichen planus is a chronic pruritic disease of the skin and mucous membranes of unknown etiology.

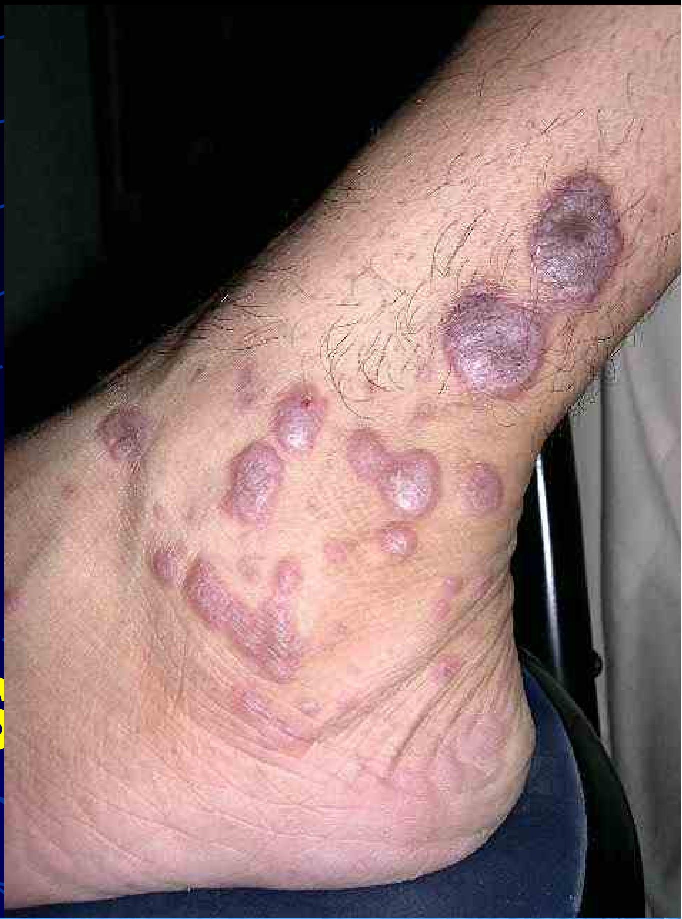
Clinical picture The pathognomonic lesions are violaceous, flat-topped, shiny, polyangular papules. The sites of predilection are the flexor aspect of wrists and forearms, lumbar region, medial thigh and around the ankles. The lesions usually heal with deep pigmentation. Mucous membrane lesions are very common, occurring in 30-70% of cases. Itching is often prominent.

Clinical varieties

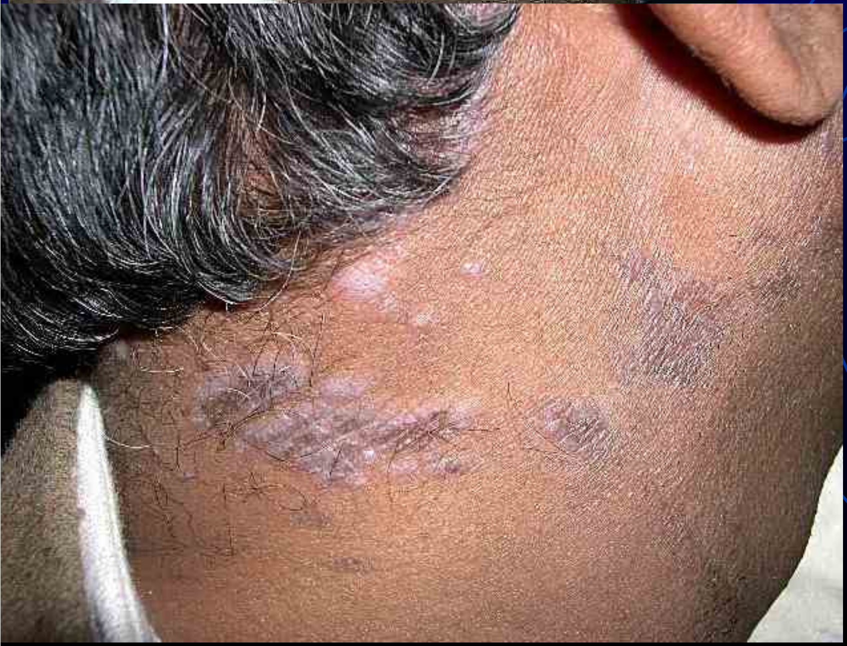
- Hypertrophic lichen planus.
- Atrophic lichen planus.
- Linear lichen planus.
- Annular lichen planus.
- Follicular lichen planus. Affects hair follicles.
- Actinic lichen planus. Affects areas exposed to sun.
- Lichen planus of the mucous membranes. The buccal mucosa and tongue are most often involved, white streaks forming lace-like are characteristic.

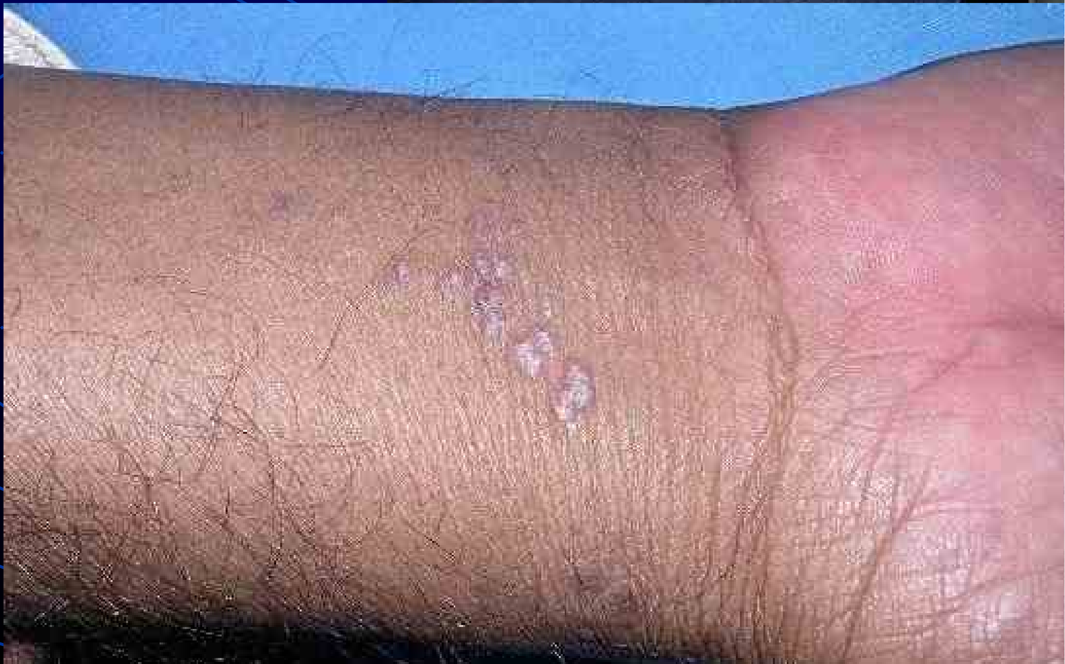
Treatment

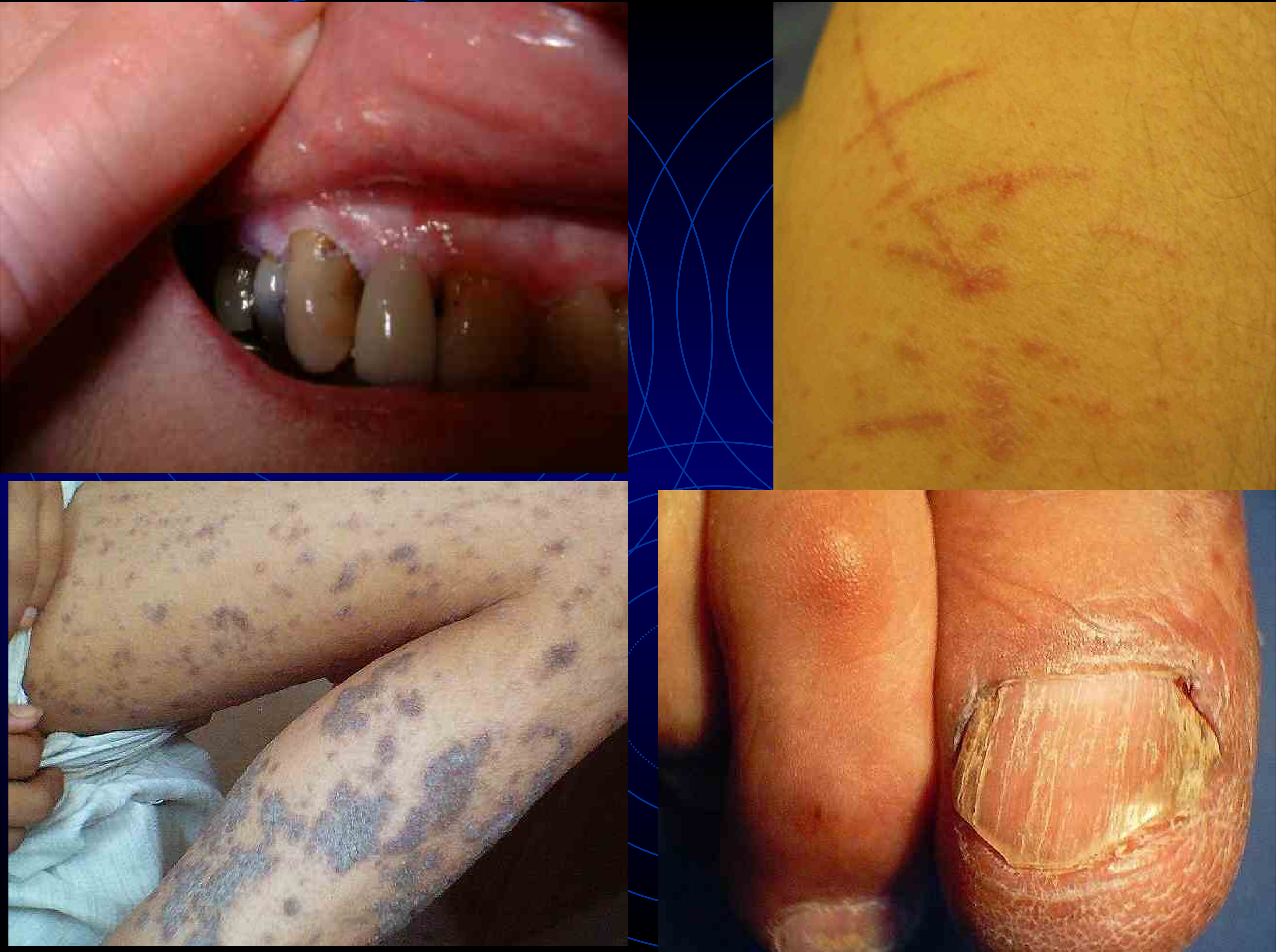




ichen planus







Pityriasis rosea

Pityriasis rosea is an acute self-limiting disease affecting children and young adults and characterized by a distinctive skin eruption. It is more common in spring and autumn. The etiology is unknown.

Clinical picture

- The first manifestation of the disease is usually the appearance of the *herald patch*, which is larger than the lesions of the later eruption and is usually situated on the trunk, thigh or upper arm. It is sharply defined, red, round or oval macule, 2-5 cm in diameter and covered by fine scales.
- After 5 to 15 days, the general eruption begins to appear in crops over a week or 10 days. The lesions have the same appearance of the herald patch. The center tends to clear, with a marginal collarette of scales. The long axes of the lesions are usually parallel to the ribs. The eruption is bilateral and symmetrical and involves the trunk and the proximal parts of the extremities. The skin lesions commonly fade after 3-6 weeks.

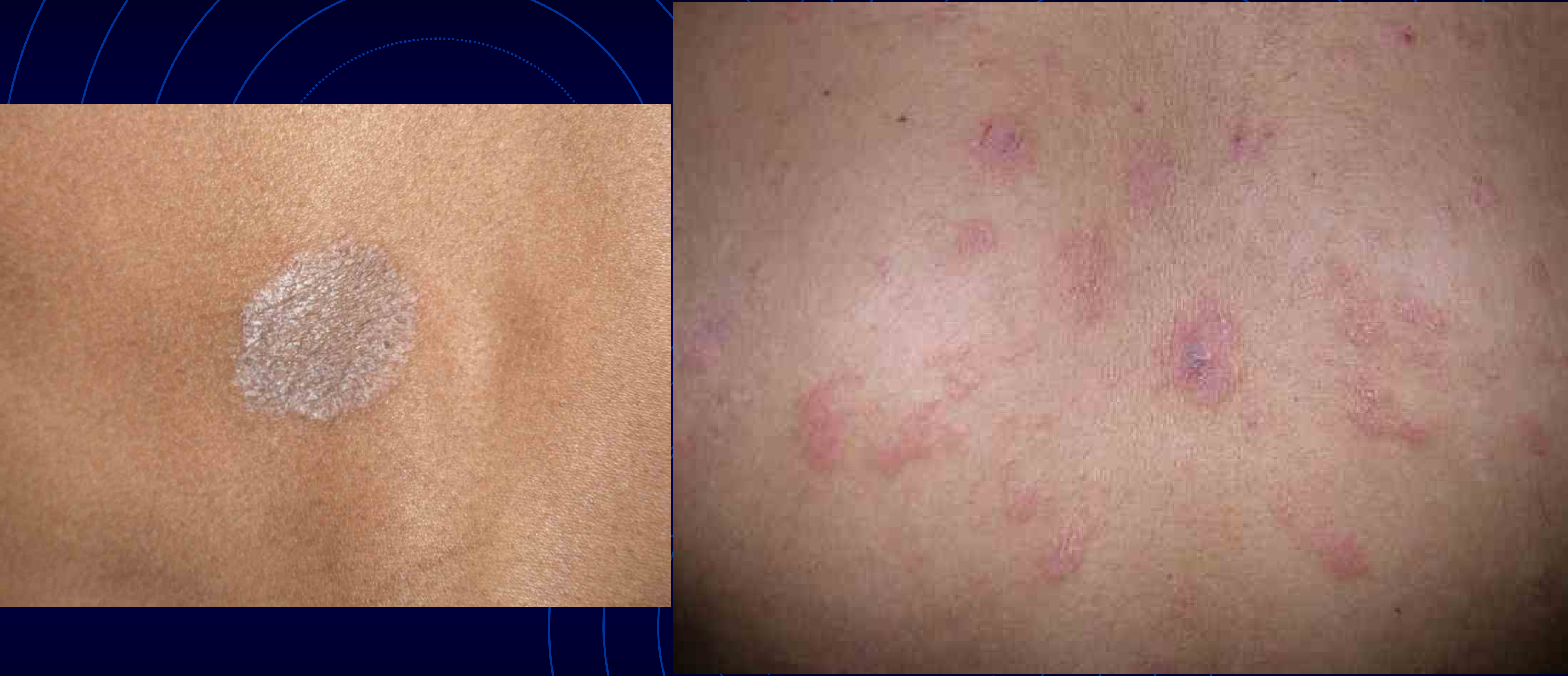
Atypical varieties

- **Inverted type.** Affects only the face and limbs.
- **Abortive type.** No lesions except the herald patch.
- **Localized type**. Confined to a single region.

Treatment

- Reassurance as the disease is self-limiting. Antihistamines for itching.

Pityriasis rosea



Pityriasis Rubra Pilaris

-It is uncommon skin disorders. Their name means that they share some of the same features, e.g. scaling (pityriasis), redness (rubra) and involvement of the hair follicles (pilaris).

-**The cause** of PRP is not known. It is not an infection and cannot be passed on to others.

-PRP occurs in individuals of all racial backgrounds and appears to affect both genders equally

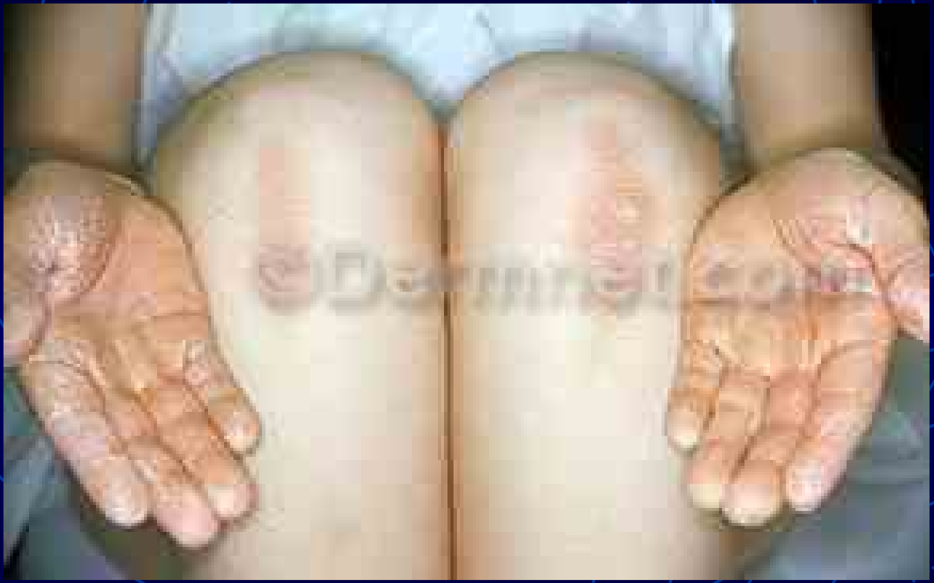
Symptoms: -The cardinal clinical features of PRP are hyperkeratotic follicular papules (plugging of the hair follicles with keratin), orange-red scaling plaques (elbows, knees, hands, feet, and ankles), scaly scalp, and palmoplantar hyperkeratosis. Well-demarcated areas of spared skin (also known as “islands of sparing”) are a frequent finding in patients with generalized involvement. Erythroderma may occur as a complication of PRP.

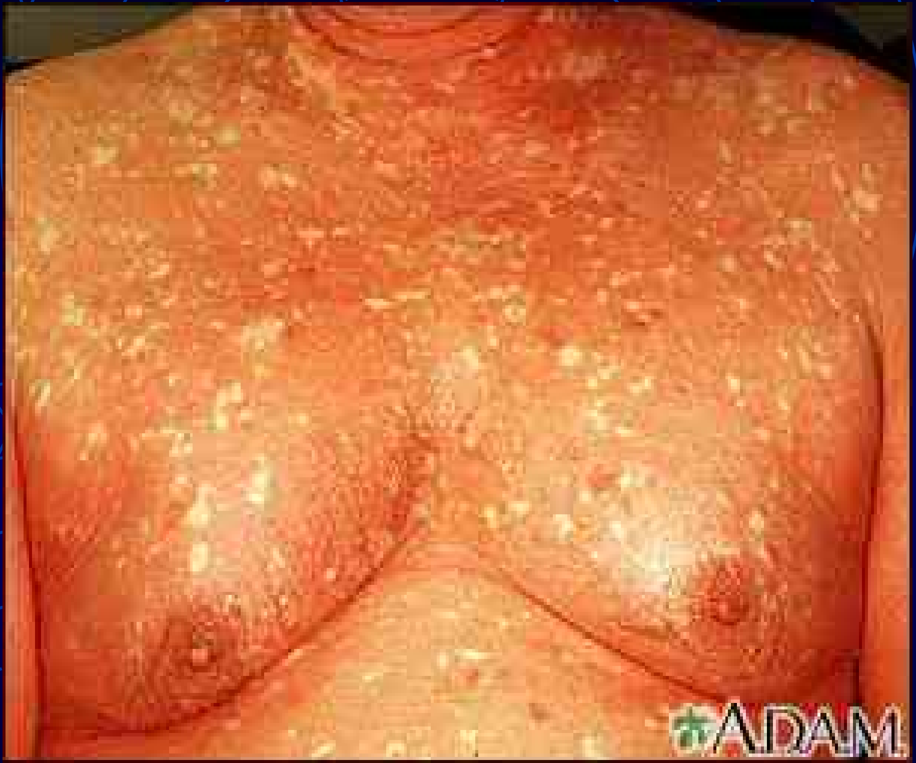
DD: psoriasis, seb. Dermatitis

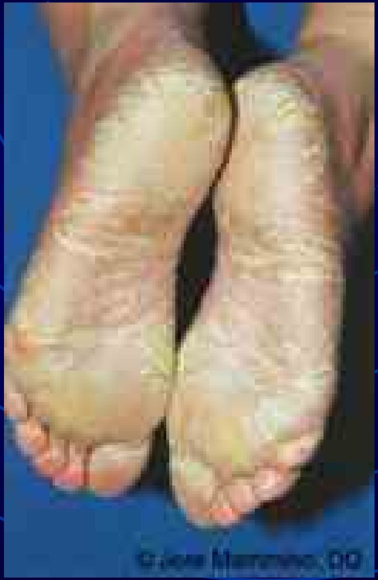
Treatment: -Steroid creams and ointments may also be used to remove the rashes.

-emollients may be used to get relive from dryness and cracking.

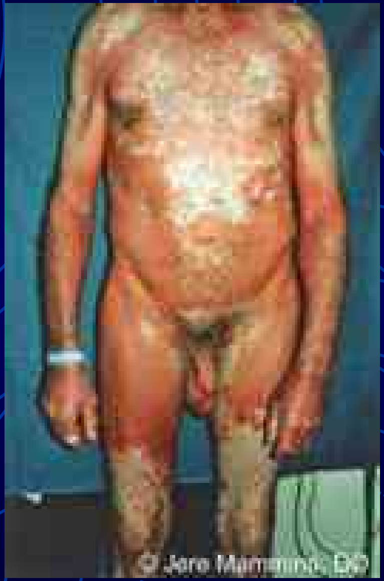
-Systemic retinoids (isotretinoin, acitretin) or methotrexate are the most commonly employed therapies


©Dermnet.com


A.D.A.M.

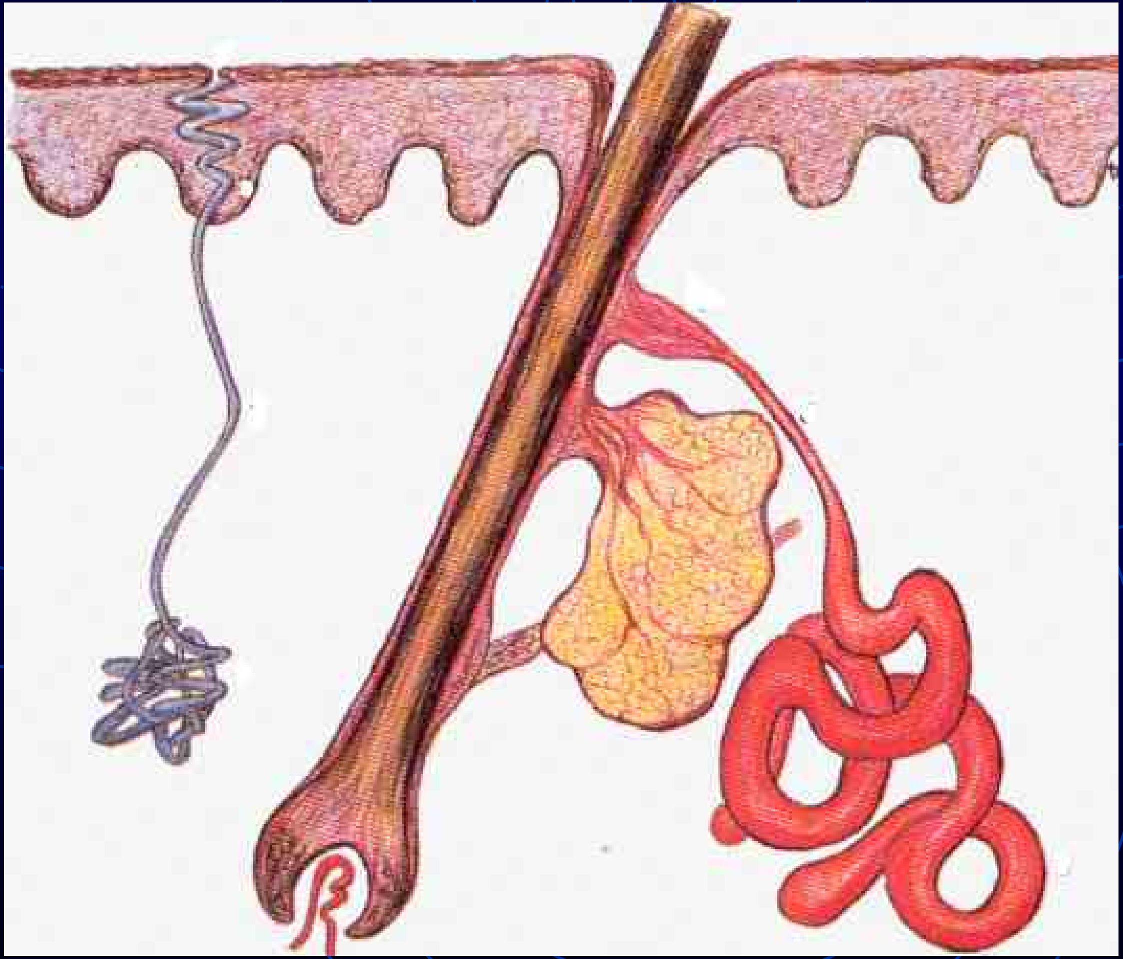


© Jere Mammino, DO



© Jere Mammino, DO

Sweat Glands Disorders



Miliaria

- *Miliaria (sweat rash) is a disease of the eccrine sweat gland caused by excessive sweating associated with obstruction of the epidermal portion of it's duct.*

- *Clinically there is two main types of miliaria according to the level of the sweat duct obstruction.*

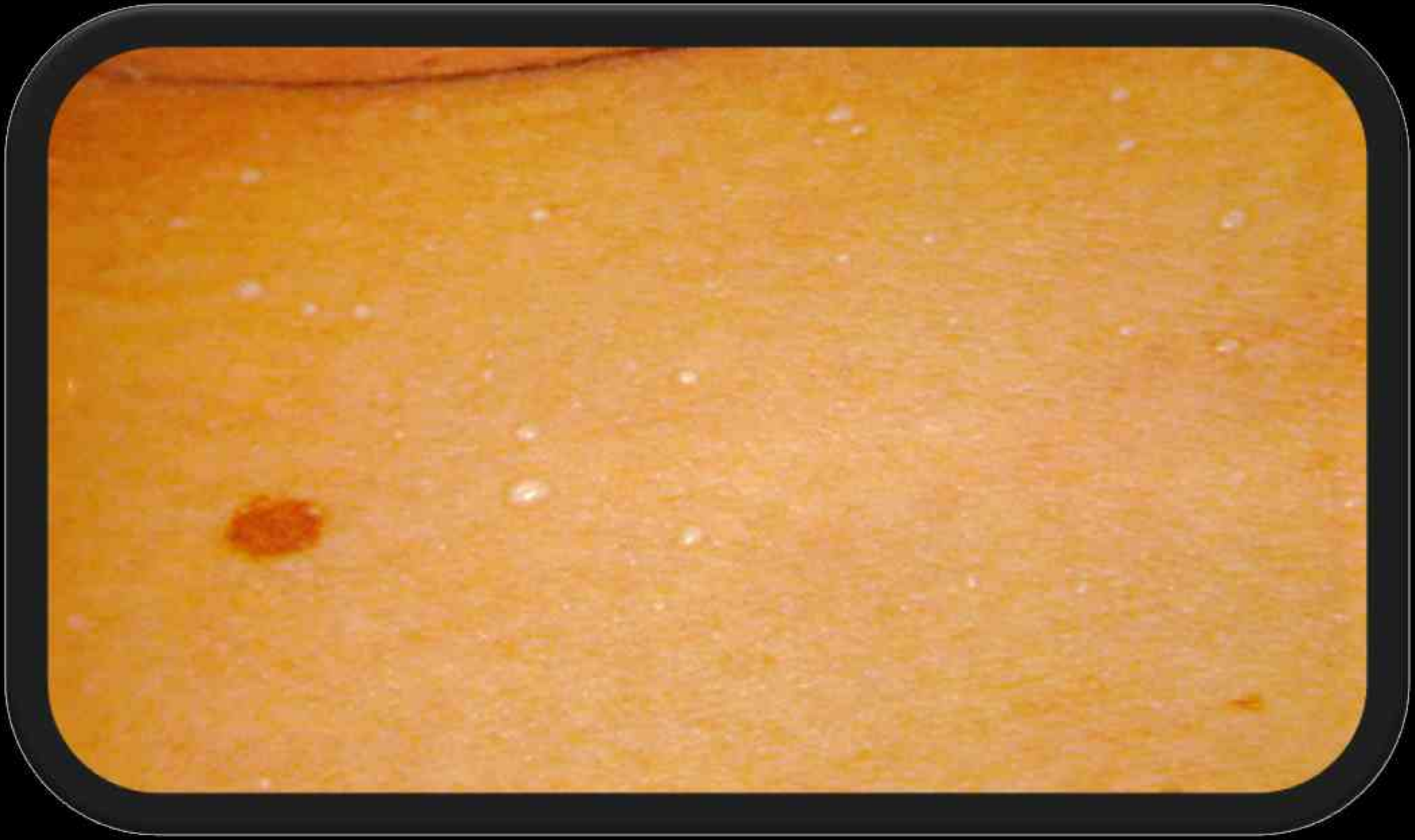
1. Miliaria Crystallina

- *The duct obstruction is in the horny layer with formation of subcorneal vesicles.*
- *It occurs with:*
  - *Sunburn.*
  - *Febrile illness associated with profuse sweating.*
  - *In newborn with excessive clothing.*

- *The vesicles are:*
  - *Non inflammatory.*
  - *Asymptomatic.*
  - *Crops of small, thin-walled vesicles with clear fluid.*

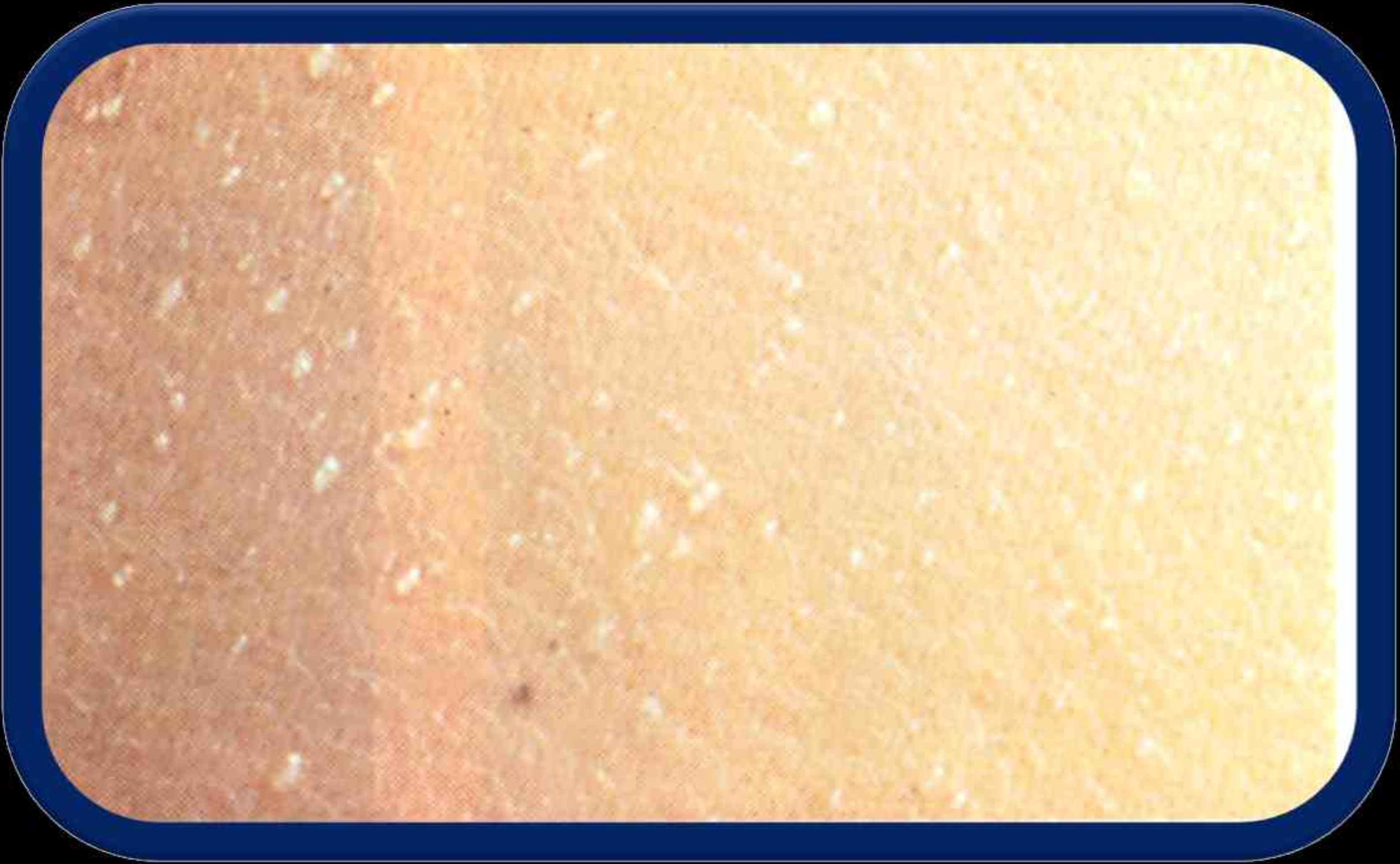
- *Mainly on the trunk.*
- *Heal with desquamation and scaling.*

Miliaria Crystallina

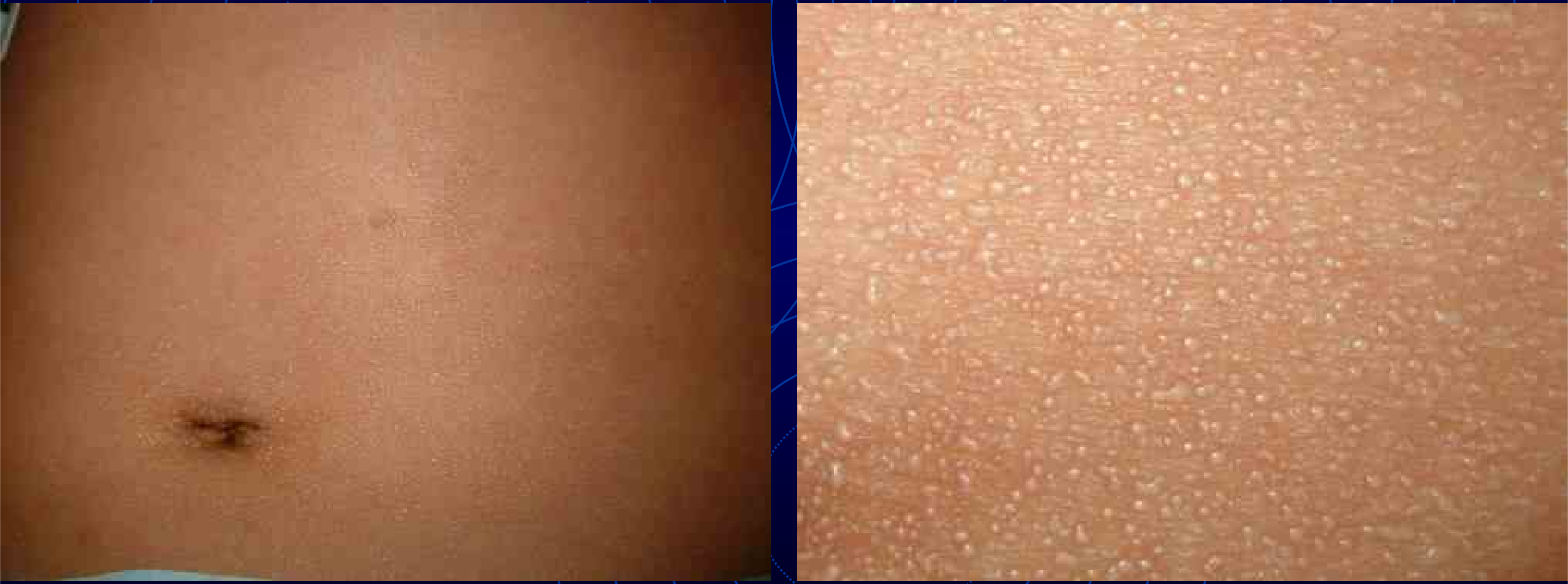


Miliaria Crystallina

Vesicles: thin-walled containing clear fluid



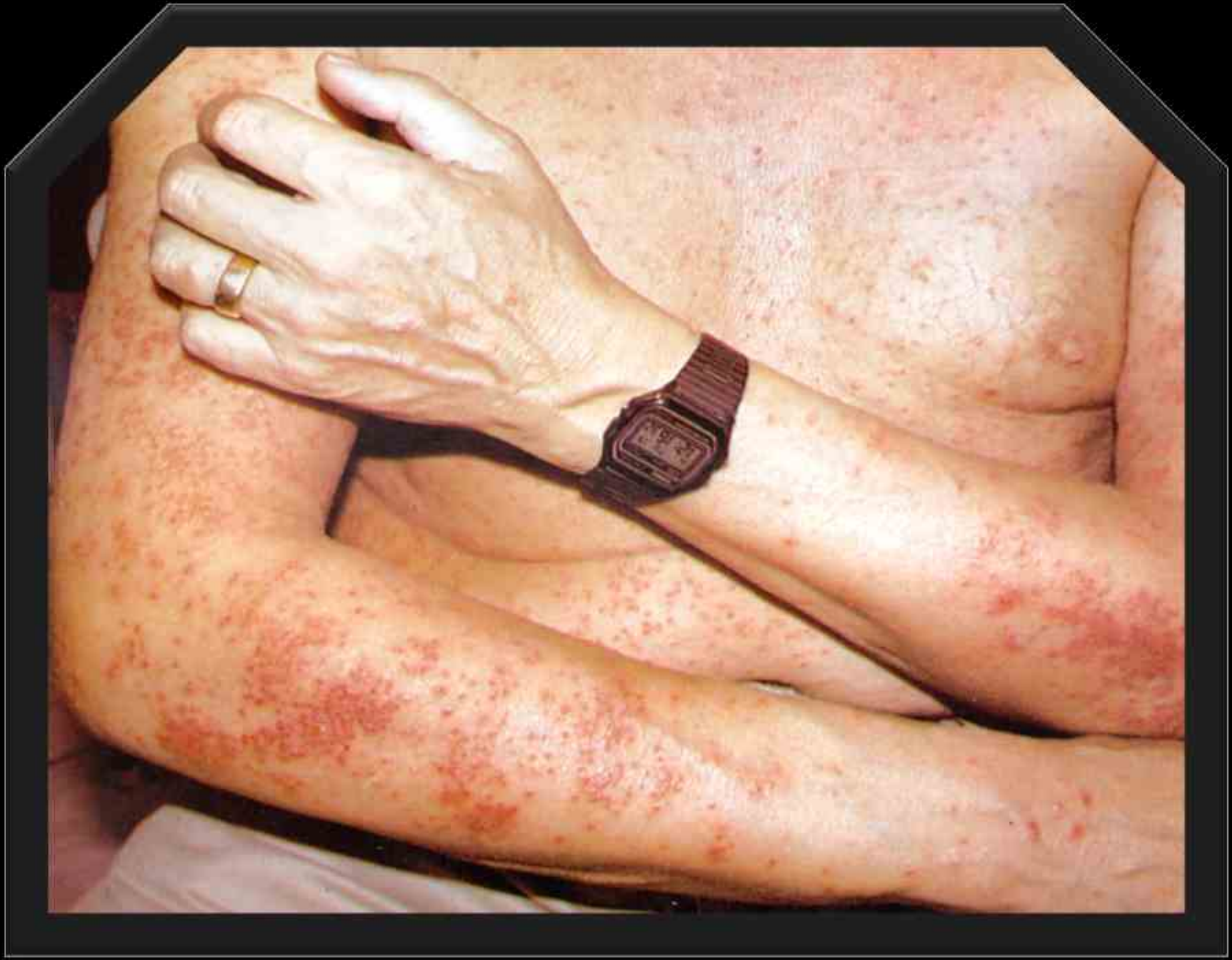
Miliaria (Sweat rash)



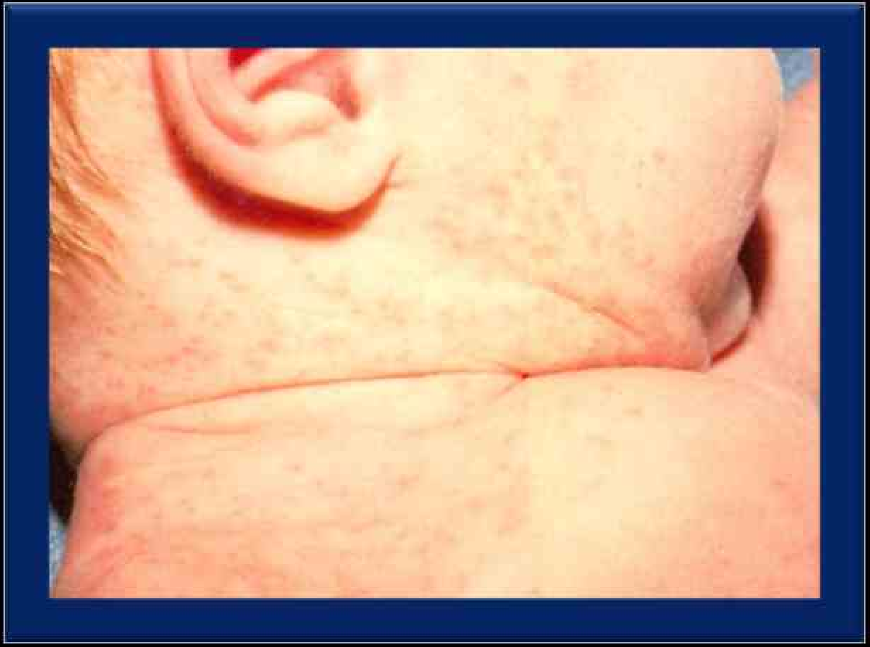
2. Miliaria Rubra (prickly heat):

- *The obstruction is in the intra-epidermal portion of the ducts.*
- *Occurs in:*
  - *Hot and humid conditions.*
  - *Areas of friction.*
  - *Flexures.*
  - *Sites of occlusion.*
  - *Lesions are:*
    - *Uniform, minute, red papules and vesicles surrounded by erythema .*
  - *In large numbers (in flexures or areas of friction with cloths).*
  - *With pricking sensation more than itching.*
  - *Can be complicated by :*
    - *Secondary infection (m. postulosa).*
    - *Or disturbance in heat regulation.*

Miliaria Rubra



Miliaria Rubra



Treatment

- The most effective treatment for miliaria is to place the patient in a cool environment.
- Avoidance of excessive clothing, excessive use of soap and contact of the skin with irritants.
- Soothing local applications e.g. calamine lotion.
- Oral antihistamines for itching and antibiotics for secondary infection.

Hyperhidrosis

Hyperhidrosis or increased sweating, may be localized to one or several areas or generalized.

Palmoplantar hyperhidrosis (emotional hyperhidrosis).

This type is usually localized to the palms and soles or axillae and may be worse during warm temperatures. It may be triggered by anxiety, stress or fear.

Gustatory hyperhidrosis.

Certain individuals regularly experience excessive sweating of the forehead, upper lip, perioral region, or sternum a few moments after eating spicy foods, tomato, chocolate, coffee, tea or hot soups.

Generalized hyperhidrosis.

It may be induced by a hot humid environment, by febrile diseases, or by vigorous excercise. Hormonal disturbances such as hyperthyroidism, diabetes mellitus and menopause may also produce generalized hyperhidrosis.

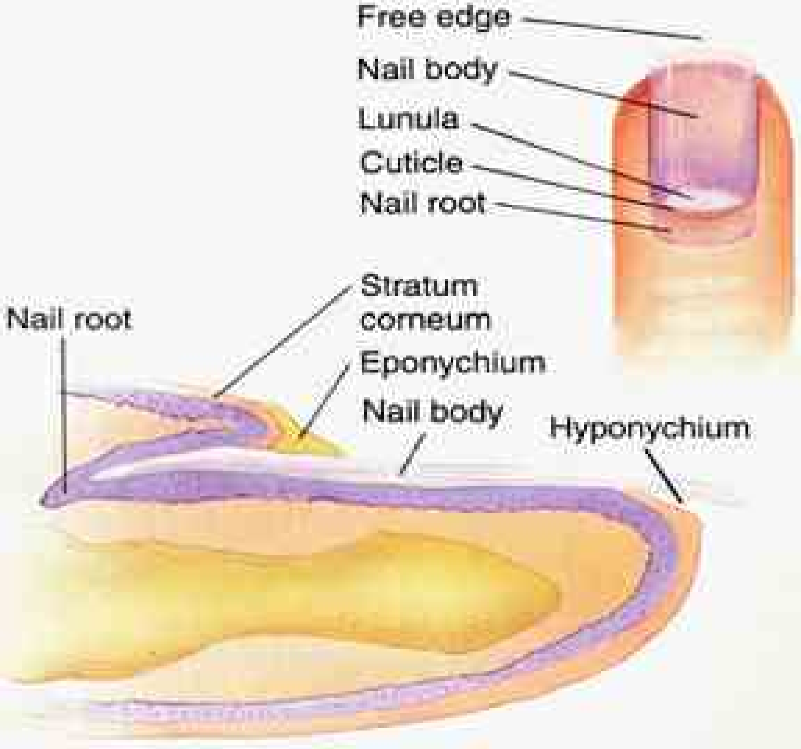
Treatment

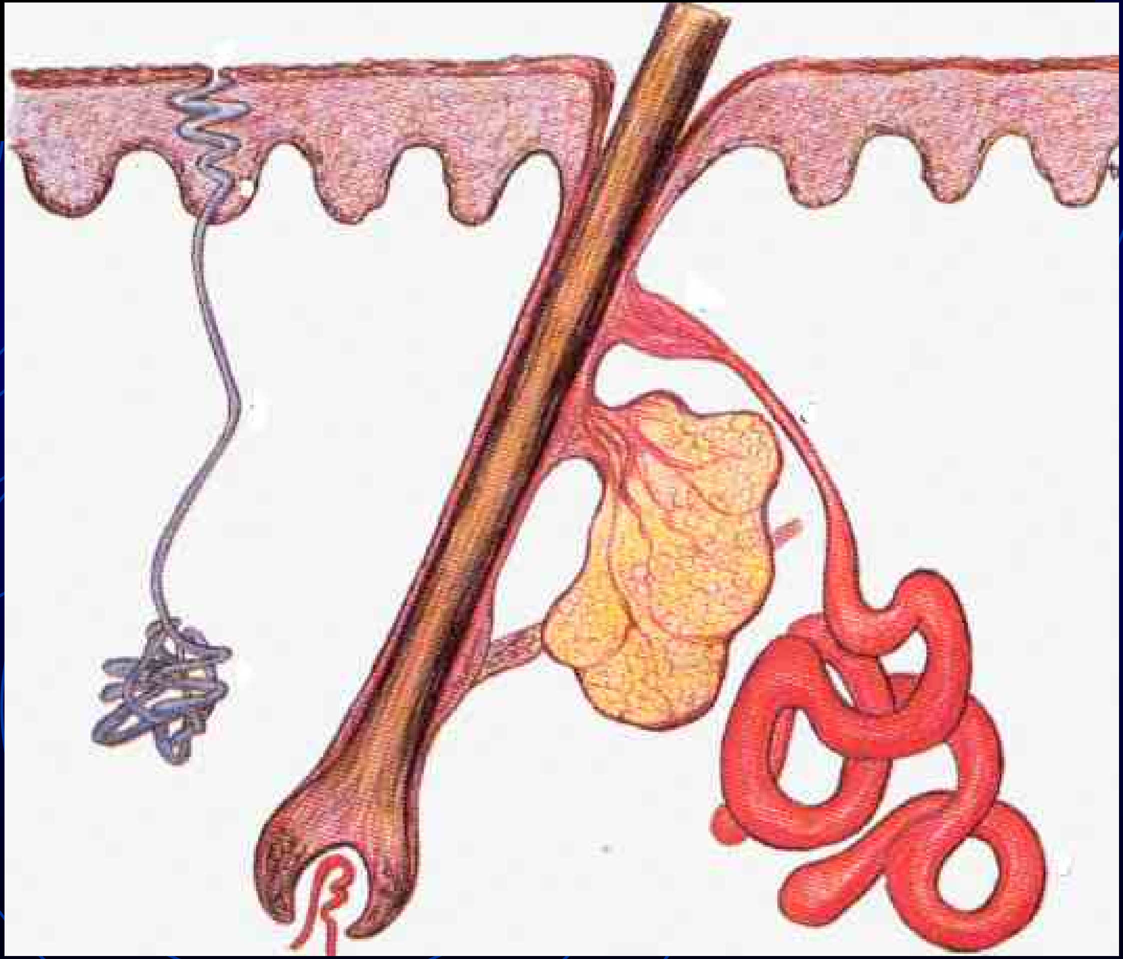
Topical aluminium chloride, anticholinergics, iontophoresis and

Nails Disorders

Skin appendages:

- Hair
- Nails
- Sebaceous glands.
- Sweet glands.

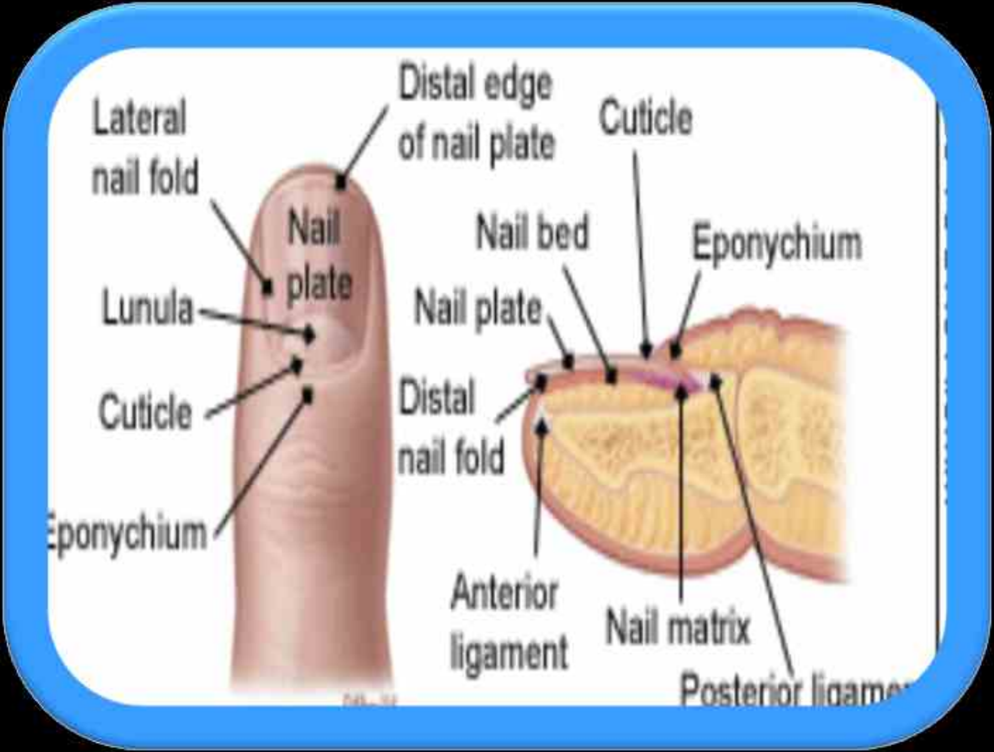

Free edge
Nail body
Lunula
Cuticle
Nail root
Nail root
Stratum corneum
Eponychium
Nail body
Hyponychium



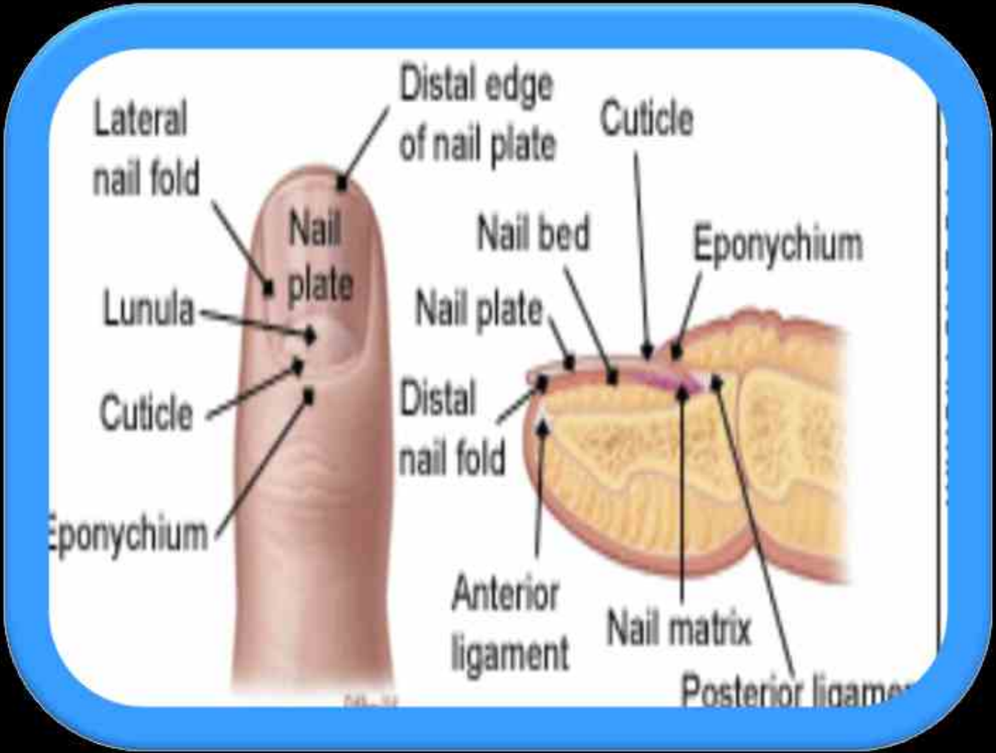
Classification of nail disorders

1. Genetic disorders: micronychia (small nails).
2. Nail infections: bacterial, fungal, viral.
3. Tumours: fibroma, melanoma.
4. Nail changes associated with skin diseases: psoriasis, lichen planus, pityriasis rubra pilaris.
5. Nail changes associated with systemic diseases.

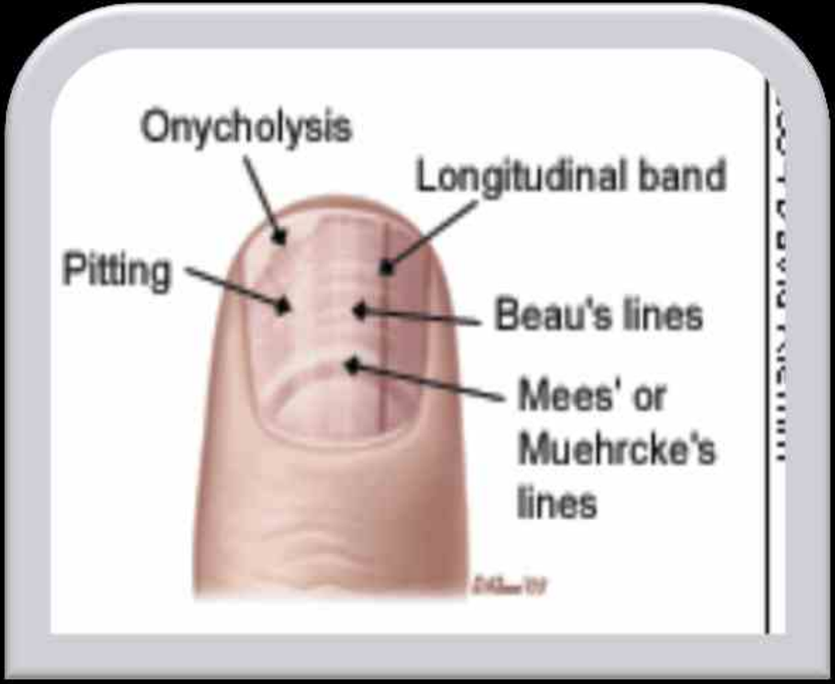
Normal Nail Structure


Lateral nail fold
Nail plate
Distal edge of nail plate
Cuticle
Nail bed
Eponychium
Lunula
Nail plate
Cuticle
Distal nail fold
Eponychium
Anterior ligament
Nail matrix
Posterior ligame

Normal Nail Structure


Lateral nail fold
Distal edge of nail plate
Cuticle
Nail plate
Nail bed
Eponychium
Lunula
Nail plate
Cuticle
Distal nail fold
Eponychium
Anterior ligament
Nail matrix
Posterior ligame

Nail Changes in Diseases


Onycholysis
Longitudinal band
Pitting
Beau's lines
Mees' or Muehrcke's lines

Beau's Line

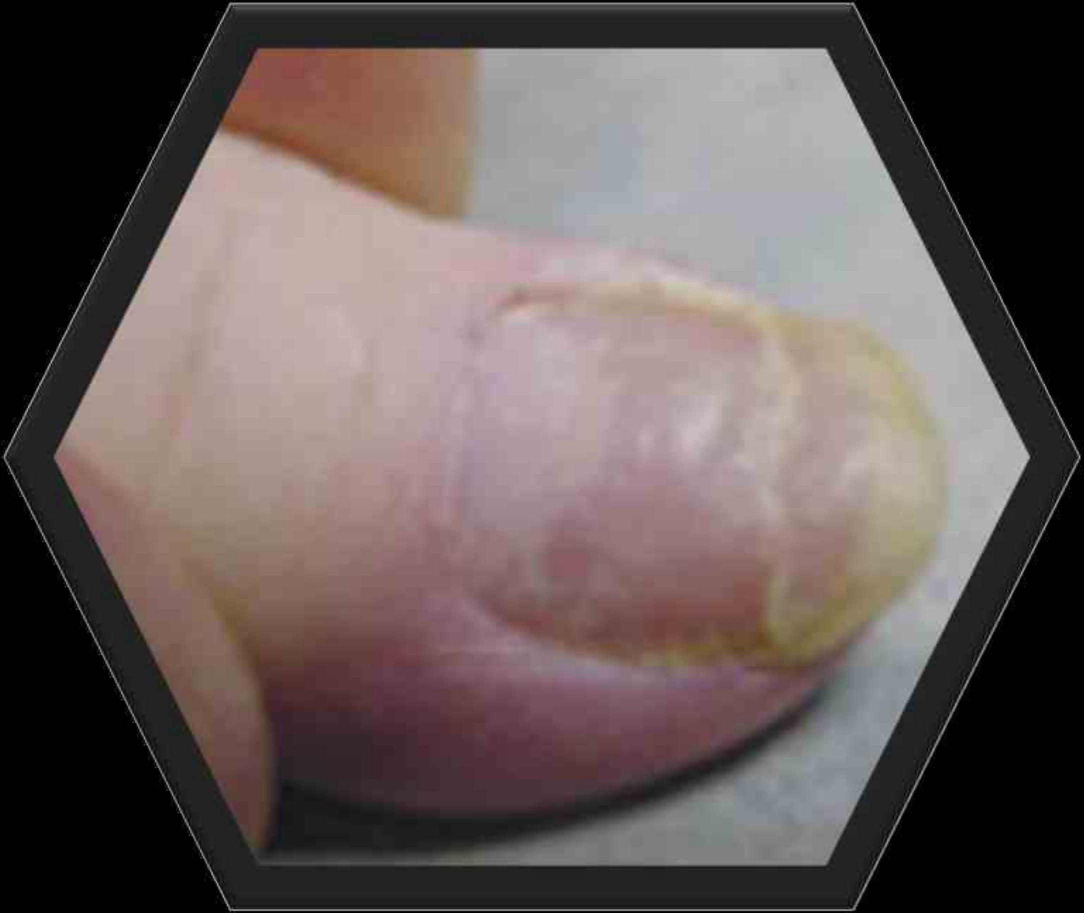
Transverse depression

"Linear transverse/horizontal groove of one or more nail plates that migrate distally from the proximal nail fold with nail growth, usually secondary to illness, trauma or malnutrition

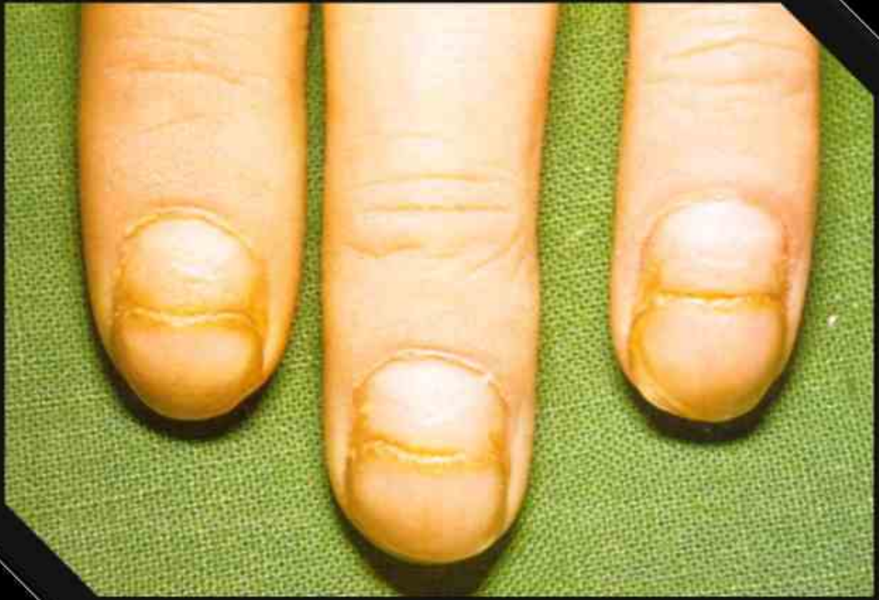
Beau's Line

Transverse depression

- DM.
- CVS.
- High fever.
- Malnutrition.
- Trauma.

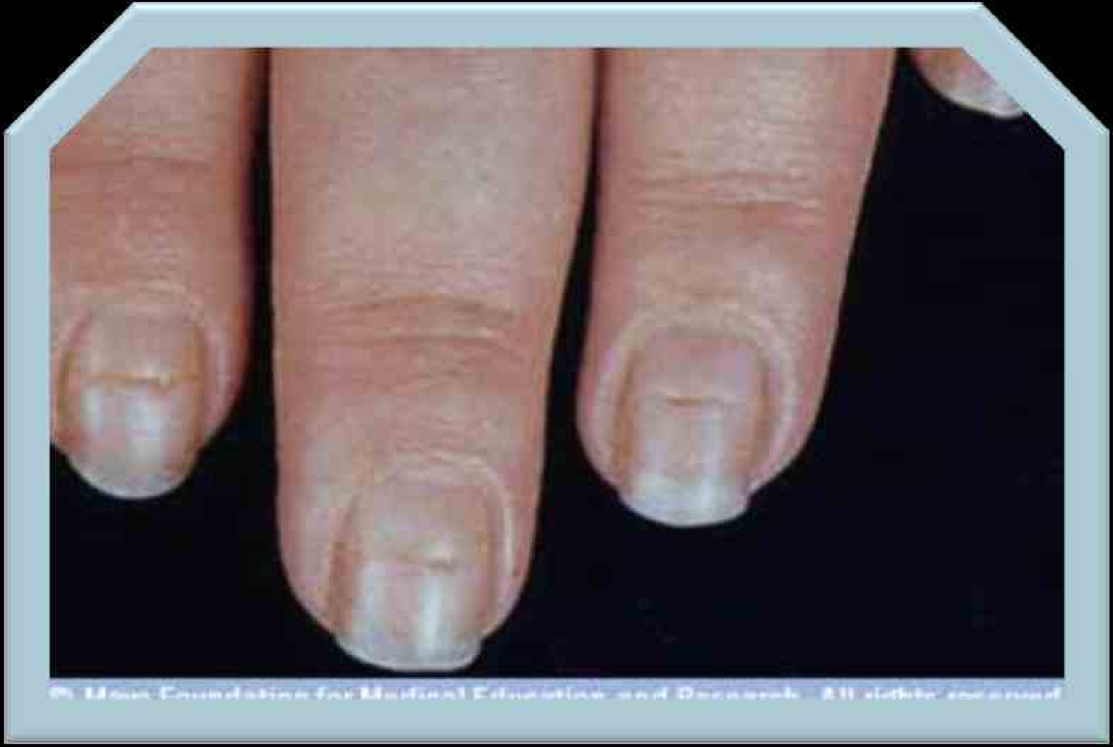


Beau’s Lines



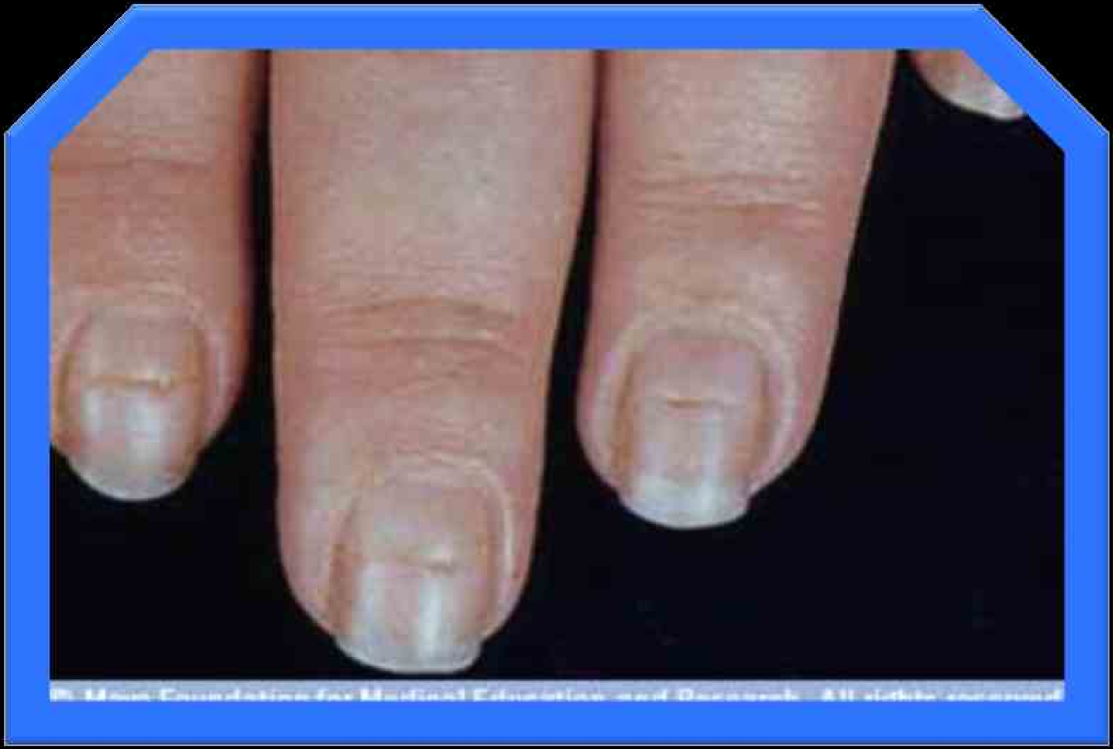
Beau's Lines

Multiple nails affected by transverse ridging



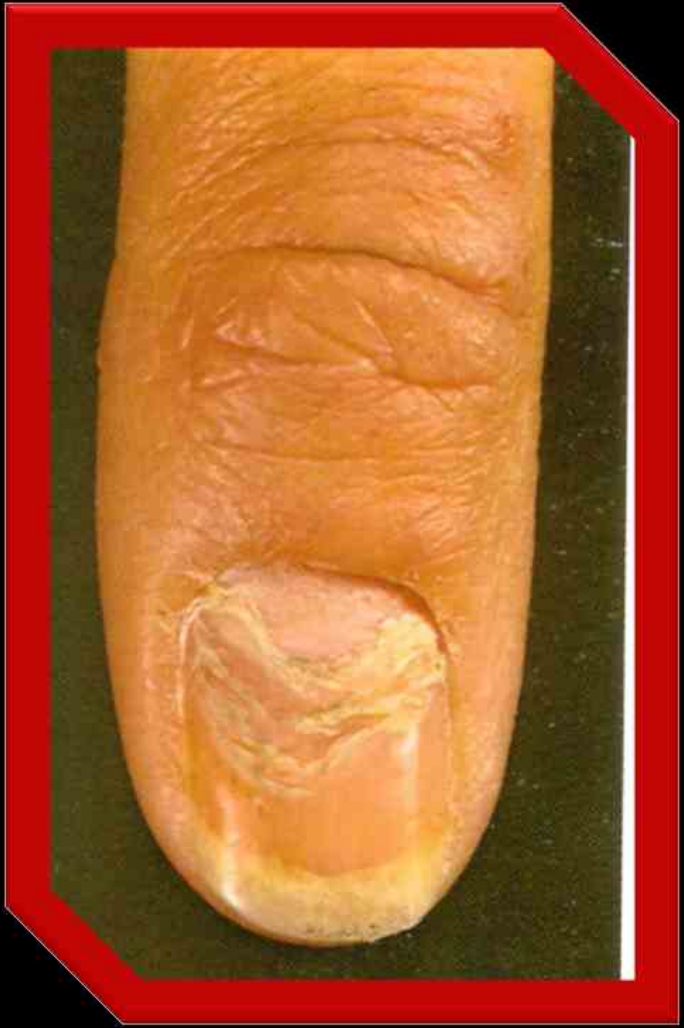
Beau's Lines

Multiple nails affected by transverse ridging

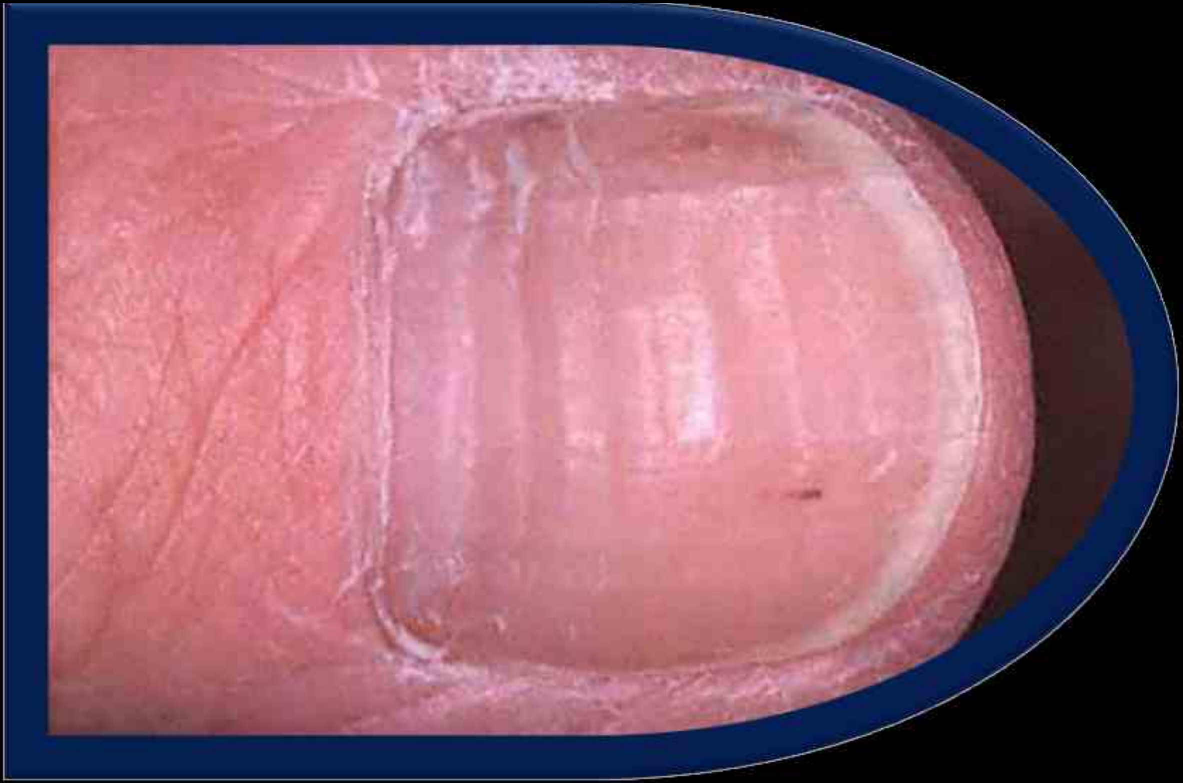


Nail Dystrophy

Eczema

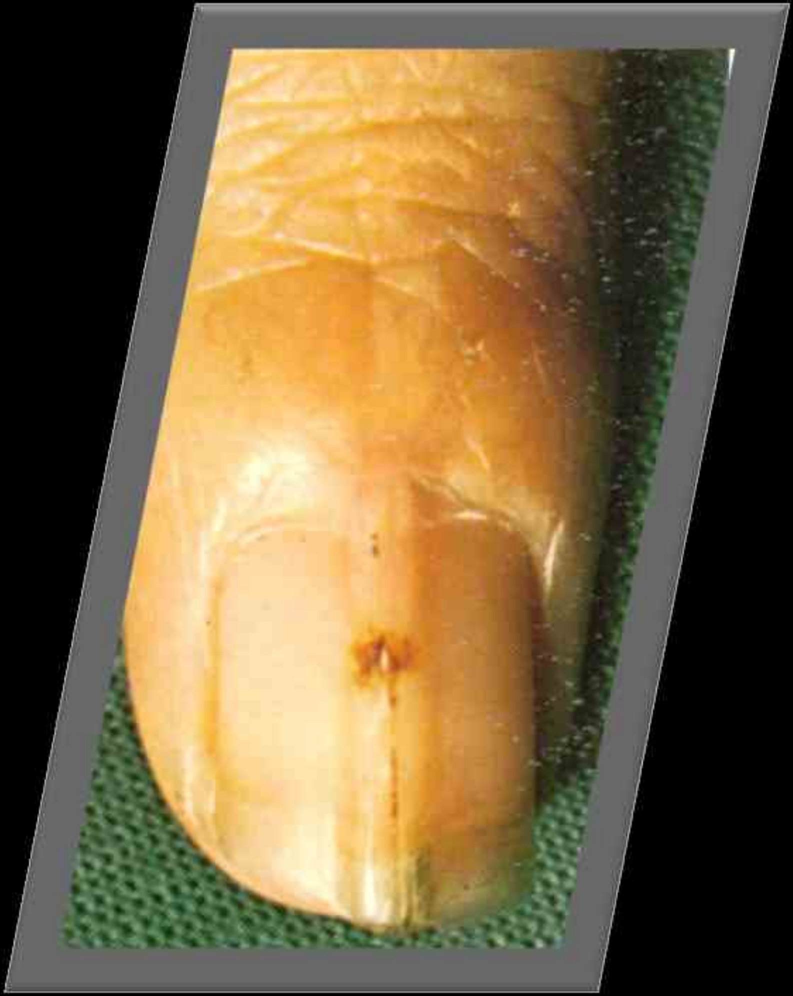


Transverse ridging of nail
Multiple lines: indicating repeated damage



- ❖ *Trauma.*
- ❖ *Febrile ill ness.*
- ❖ *Drugs.*

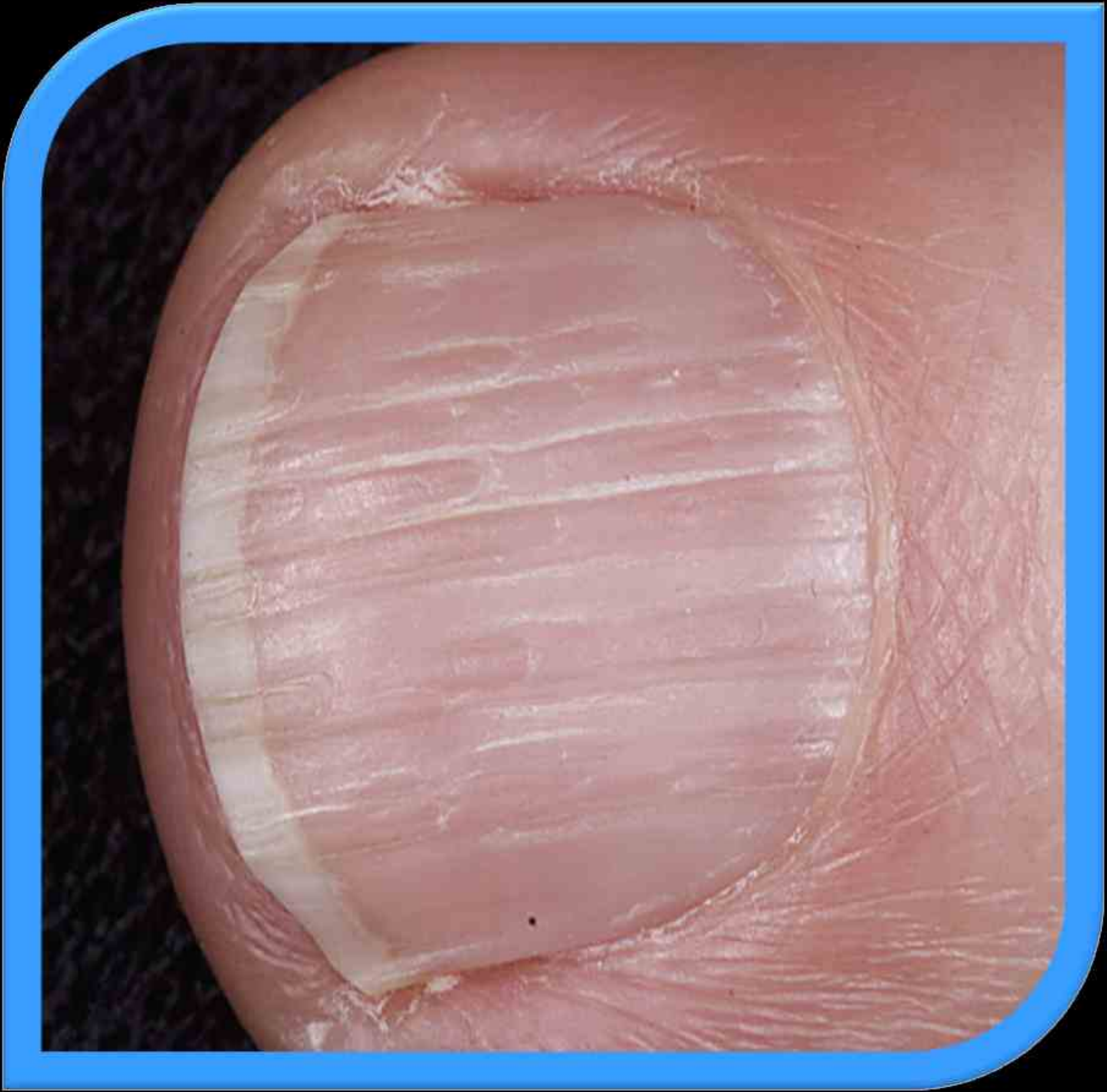
Traumatic Nail Dystrophy



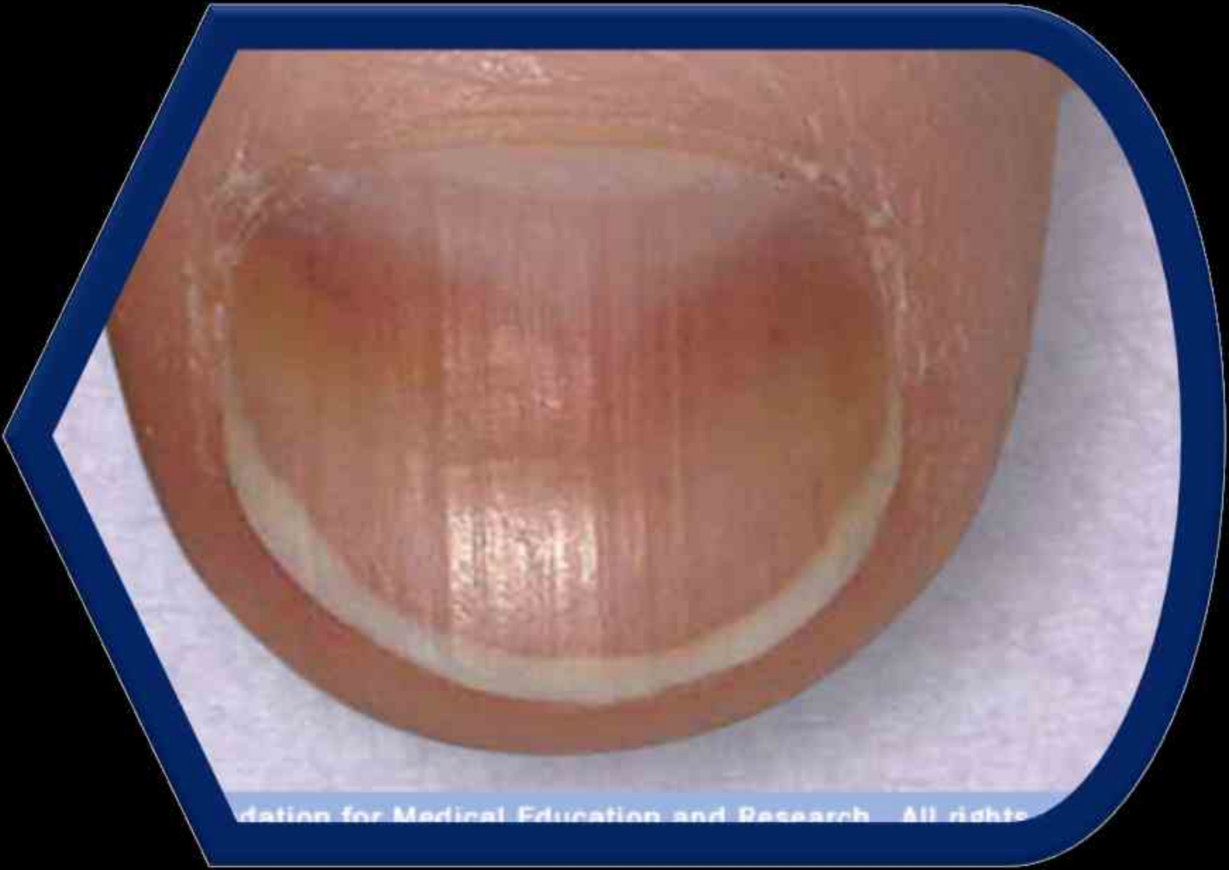
Longitudinal Ridging

Diffuse nail matrix damage

- Skin diseases.
- Vascular causes.
- Trauma.
- Tumors.
- Aging.



Longitudinal Ridging

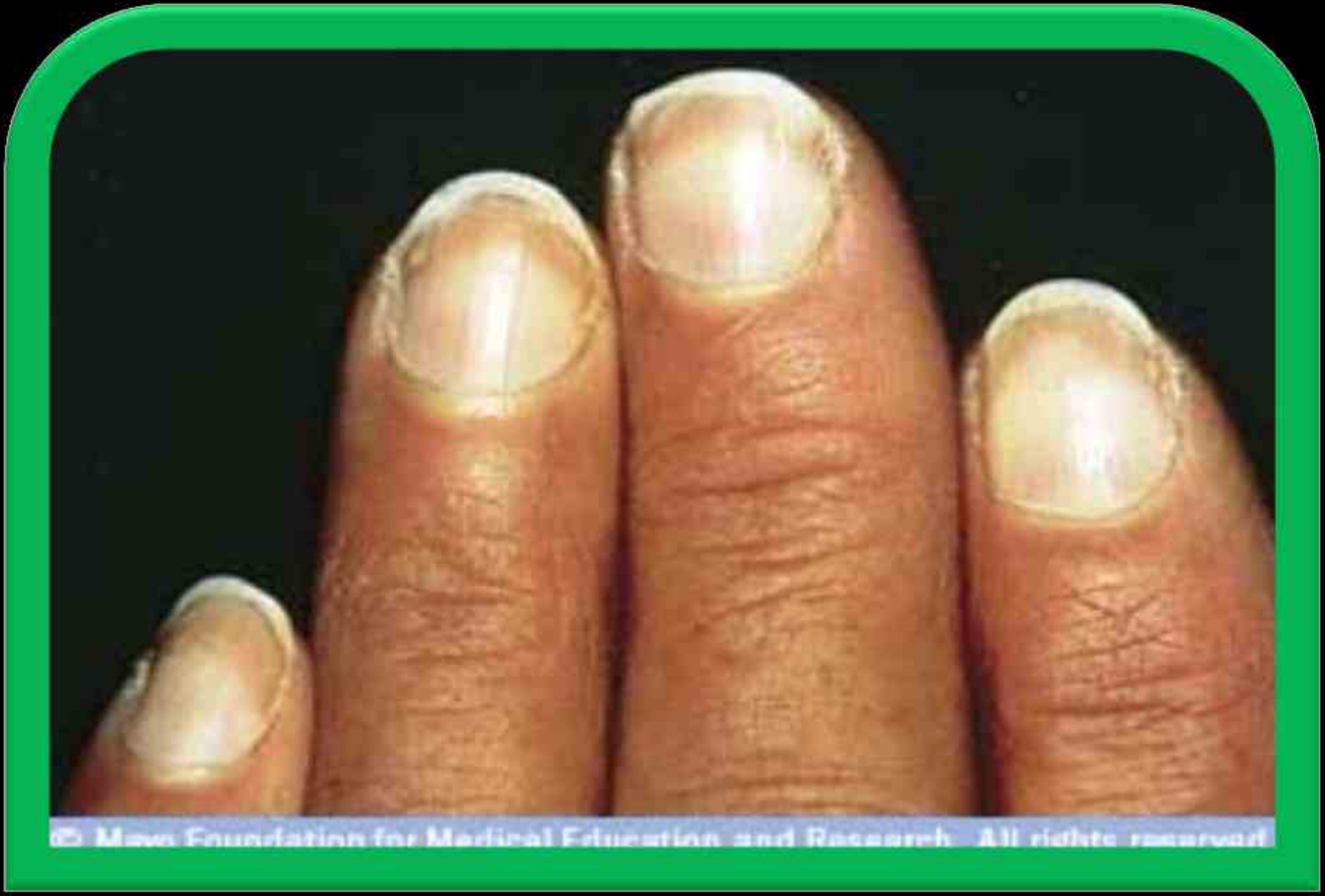


dation for Medical Education and Research. All rights

Half and Half Nails

Chronic Renal Failure

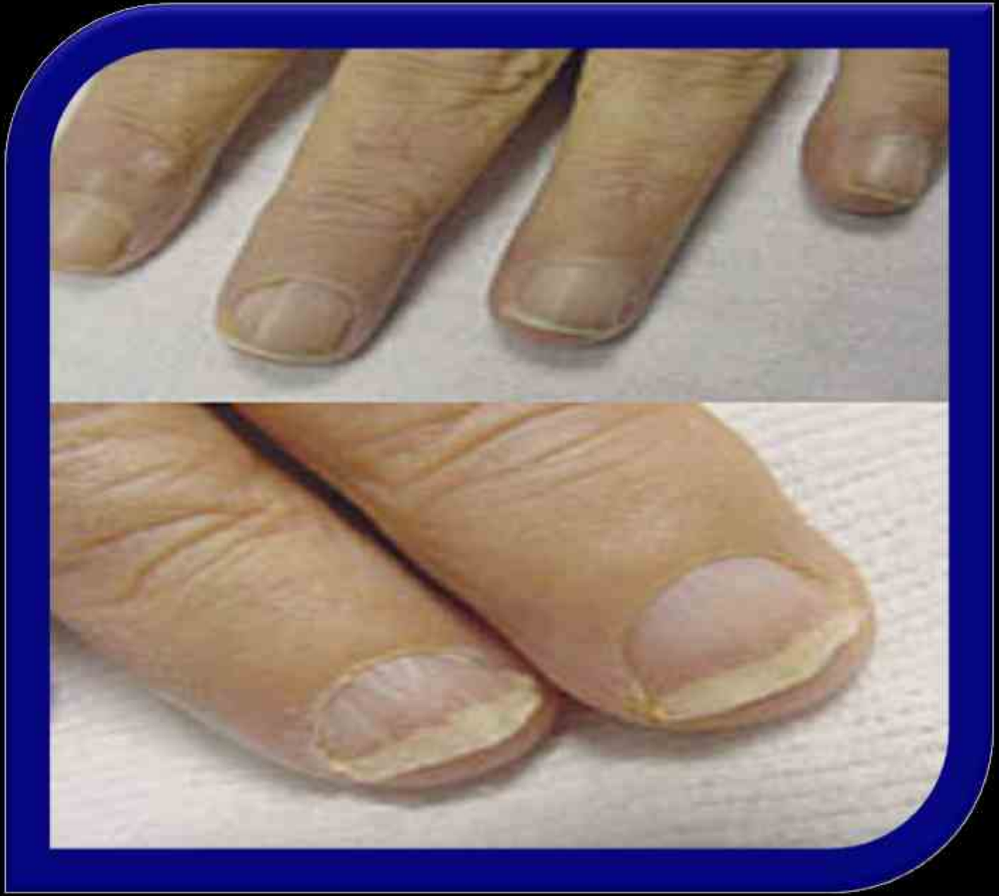
Proximally white and distally pink



© Mayo Foundation for Medical Education and Research. All rights reserved.

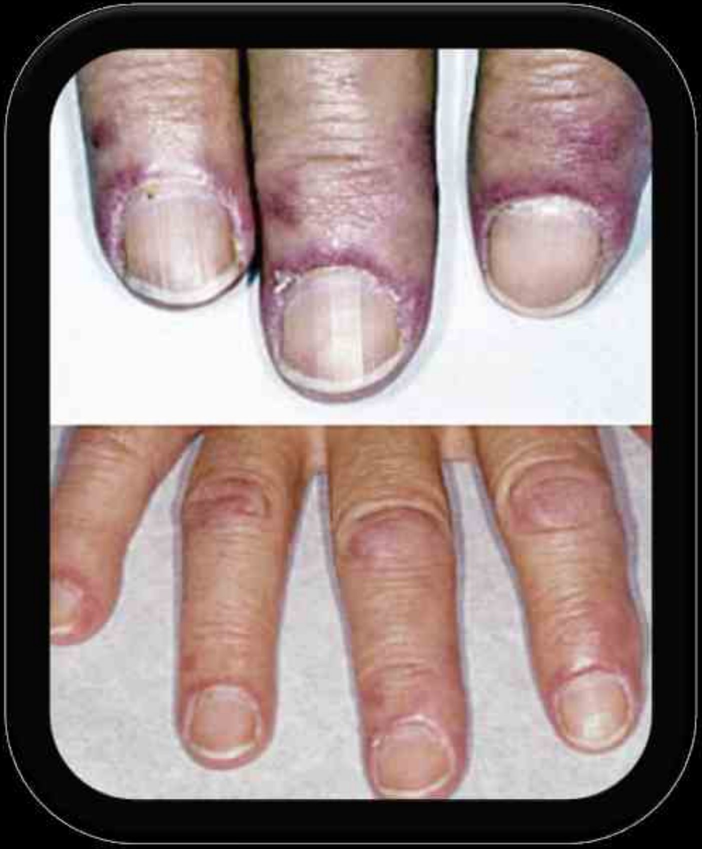
Half and Half Nails

Chronic Renal Failure



Peri-ungual Telangiectasia

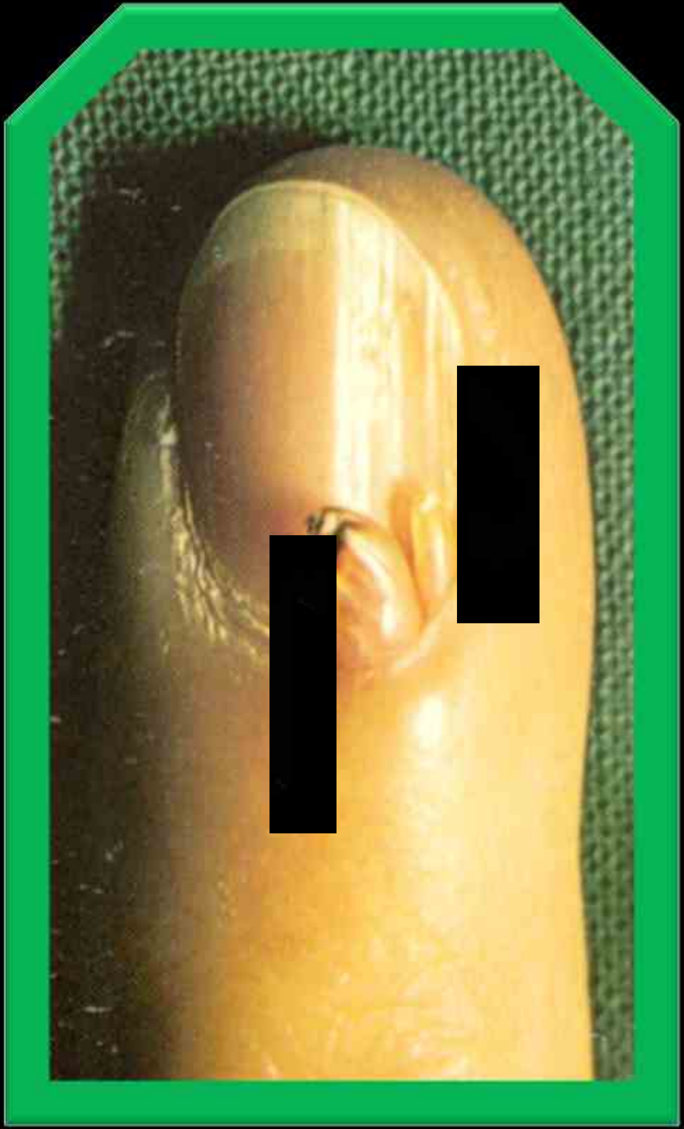
Connective tissue diseases e.g. SLE & DM



Dermatomyositis

Telangiectasia of the nail fold and papules

Peri - Ungual Fibromas

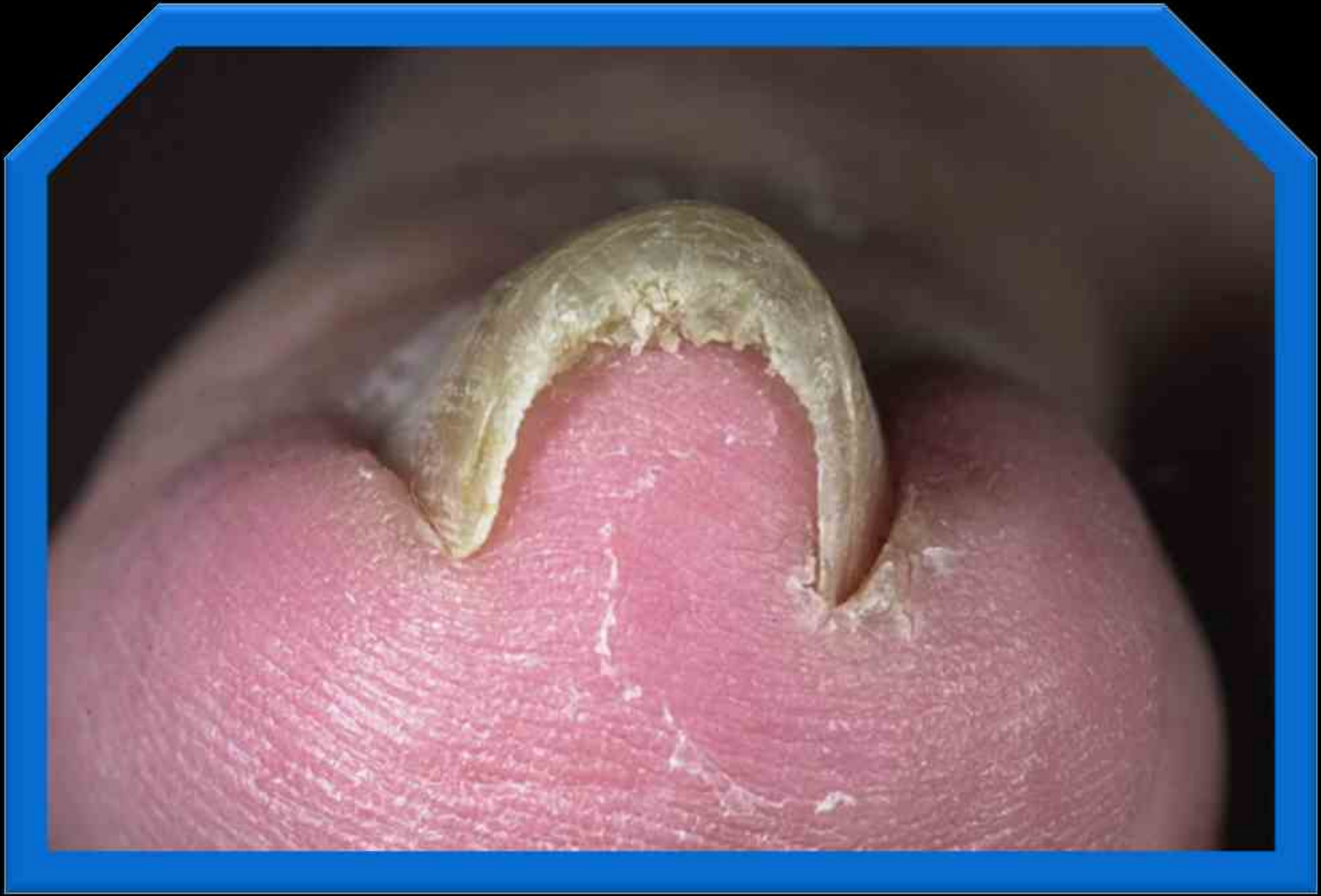


[redacted]

[redacted]

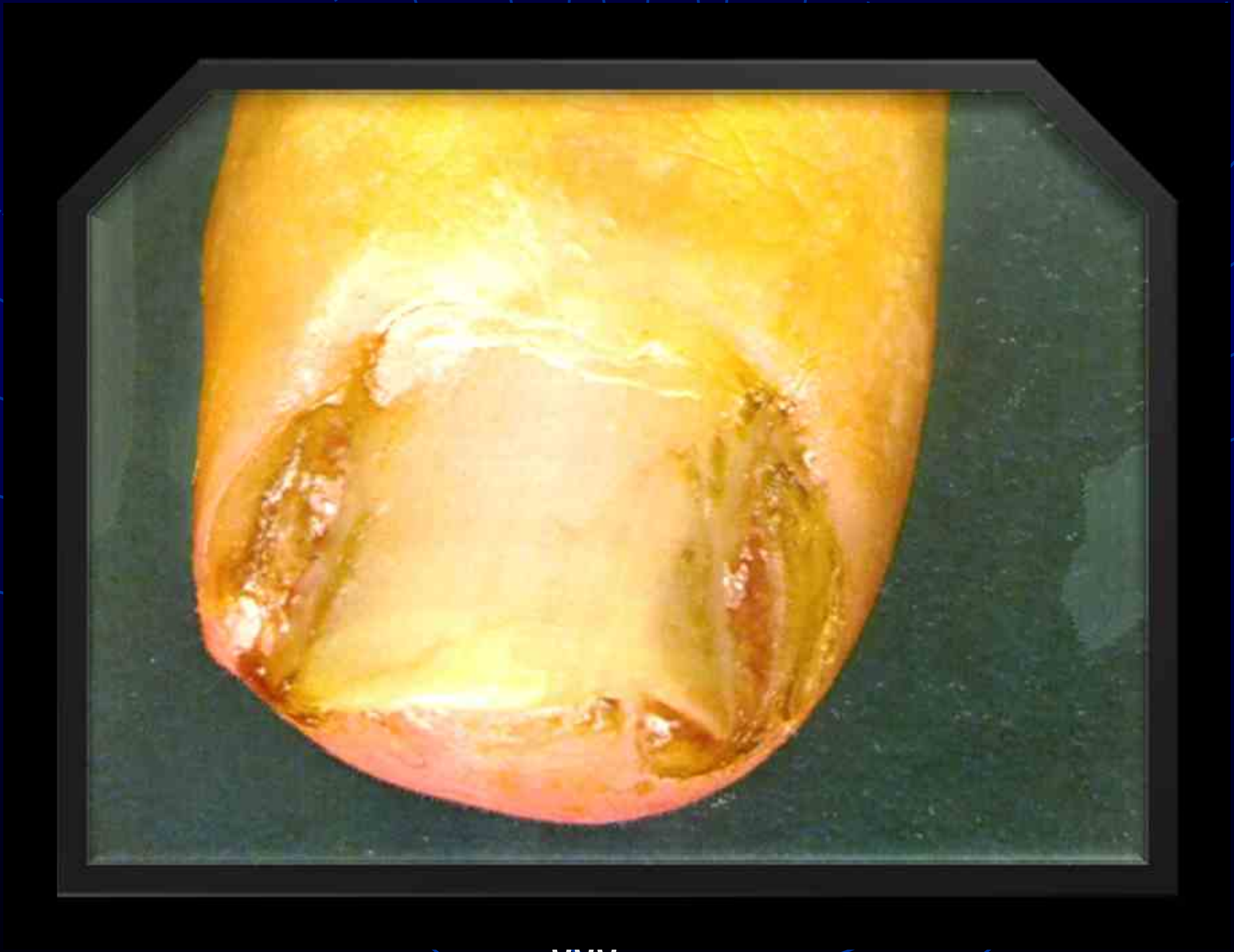
Pincer Nail

Inherited or due to trauma or B-blockers



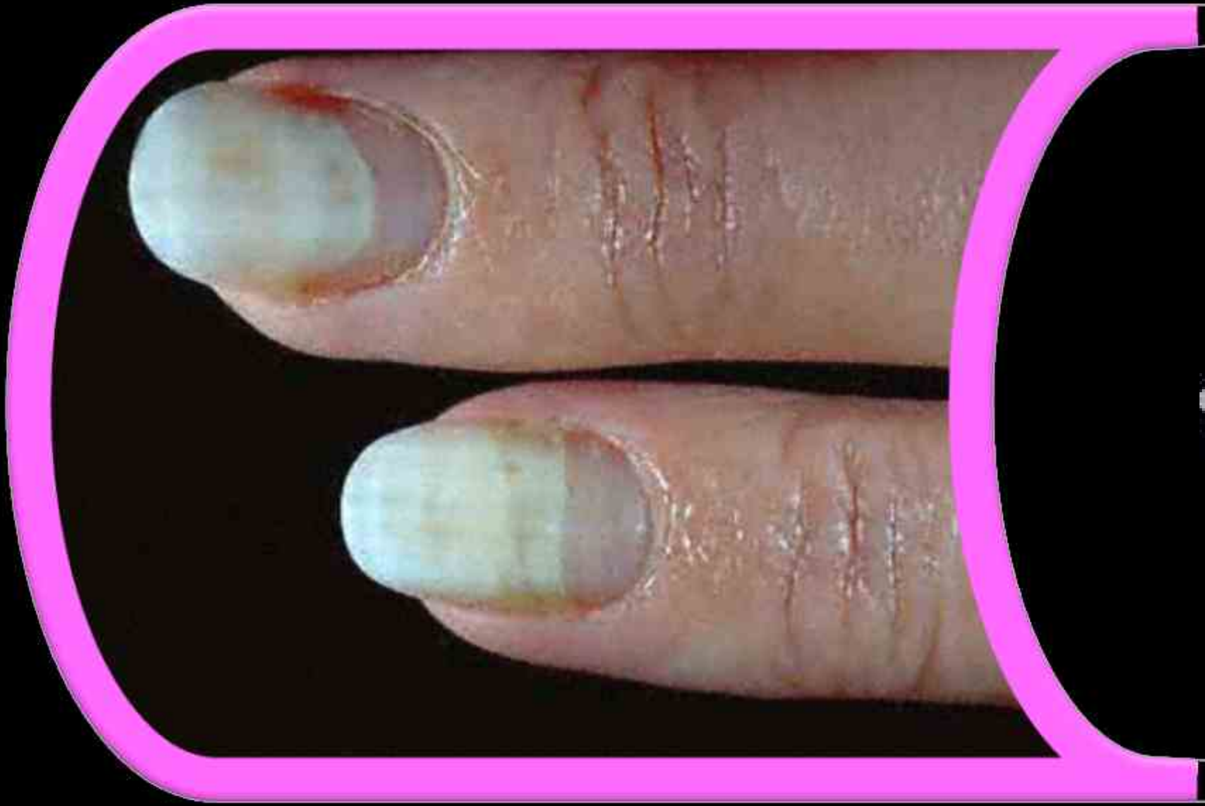
Ingrowing Nail

Big toe



Onycholysis

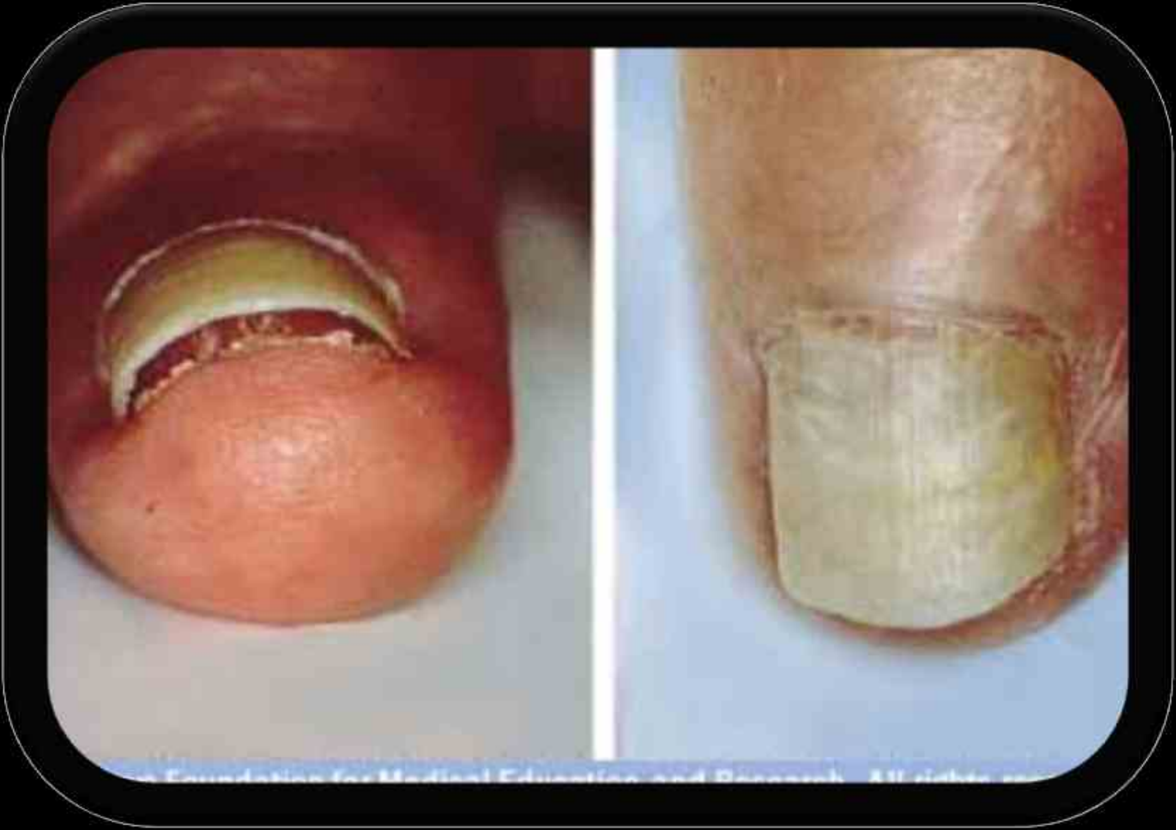
Separation of nail plate from its bed



- *Trauma.*
- *Infection.*
- *Thyroid Ds.*
- *Drugs.*
- *Skin Ds.*

Onycholysis

Endocrine Ds: Hypo-and-hyperthyroidism



Foundation for Medical Education and Research. All rights re

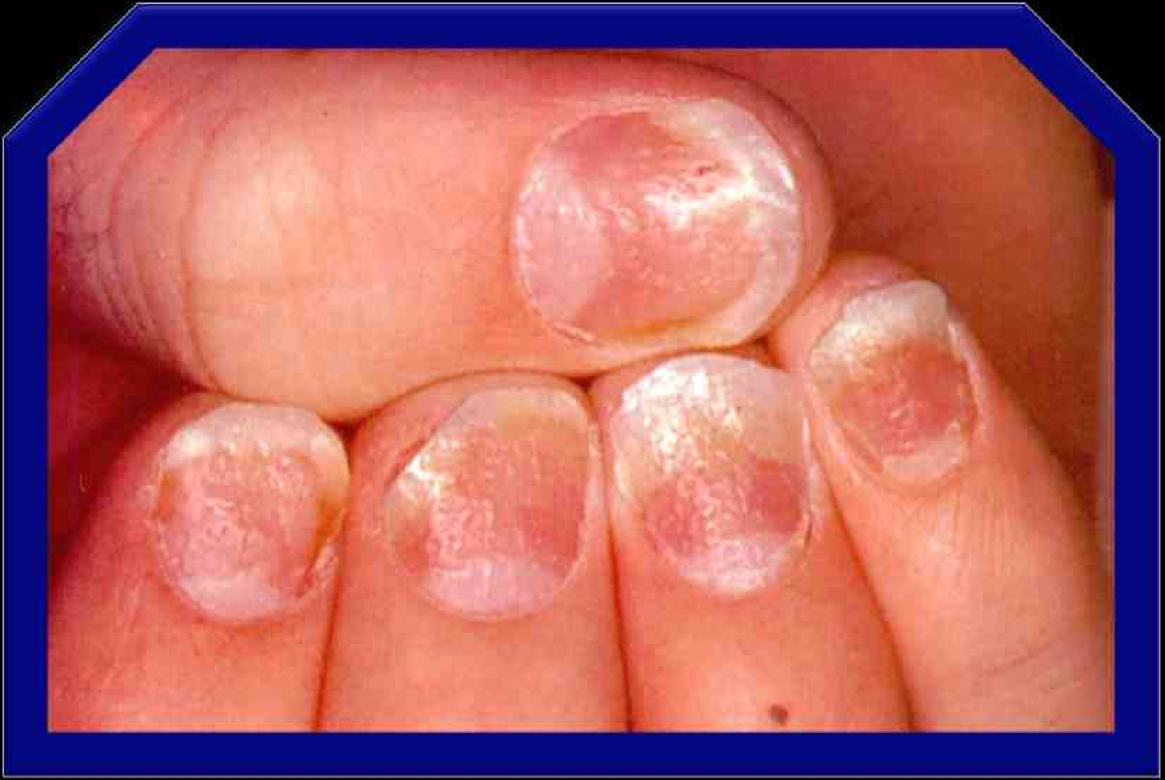
Complete separation of nail from its bed

Pitting of nails

Psoriasis

AA

Eczema

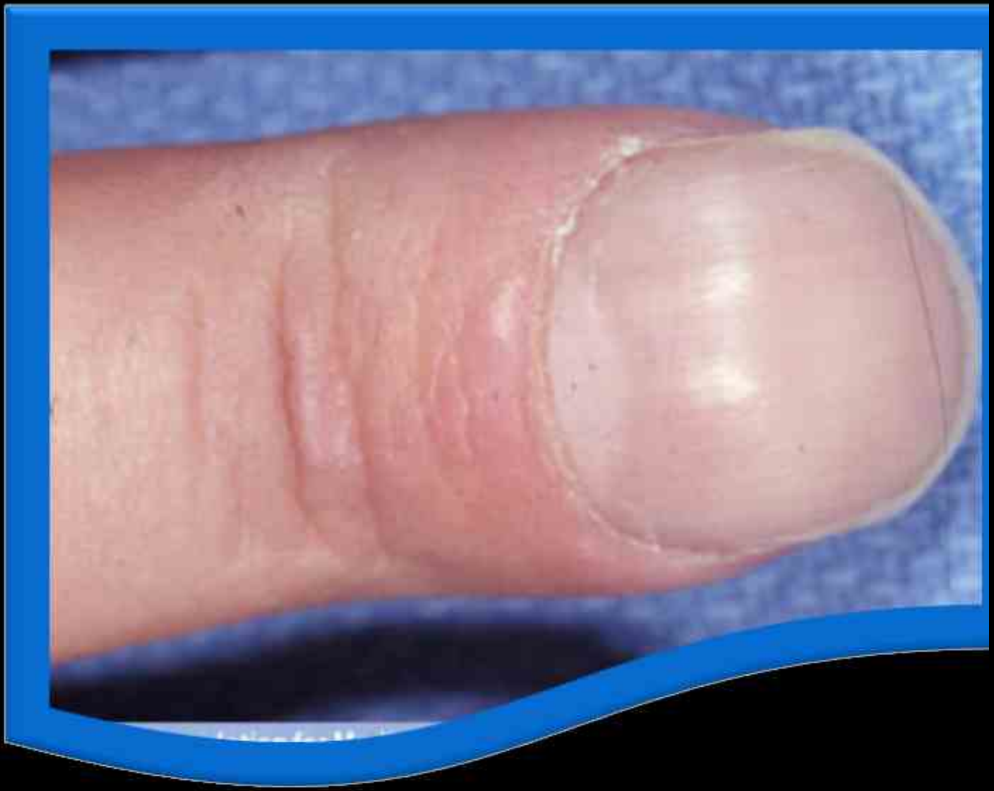


- ***Clubbing:***
  - Curvature of the nail plate with loss of the normal angle between the nail plate and the proximal nail fold, occurs in:
    - Pulmonary and mediastinal diseases.
    - Cyanotic cardiovascular diseases.
    - Hepatic cirrhosis.
- ***Koilonychia:***
  - Concavity of the nail plate (spoon nail) occurs in:
    - Iron deficiency anemia.
    - Psoriasis.
    - Diabetes.

Definitions of nail changes

- *Onycholysis:*
  - Separation of nail plate from the nail bed, occurs in:
    - Trauma.
    - Cosmetics.
    - Psoriasis.
    - Hypo and hyperthyroidism.
    - Drugs.
    - Onychomycosis.
- *Pitting:*
  - Shallow depression in the nail plate, occurs in:
    - Psoriasis.
    - Alopecia areata.
    - Eczema.
    - Trauma.

Clubbing of finger and nail

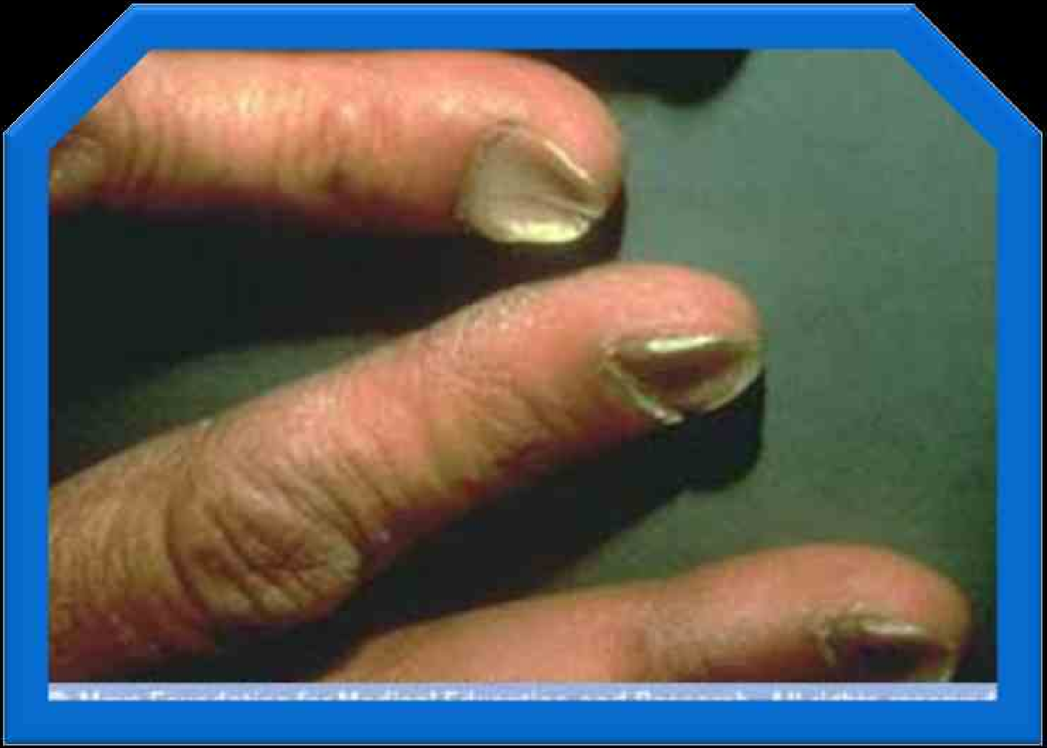


- Cardiovascular.
- Bronchopulmonary.
- Gastrointestinal.
- Infection.
- Endocrine .

Koilonychias

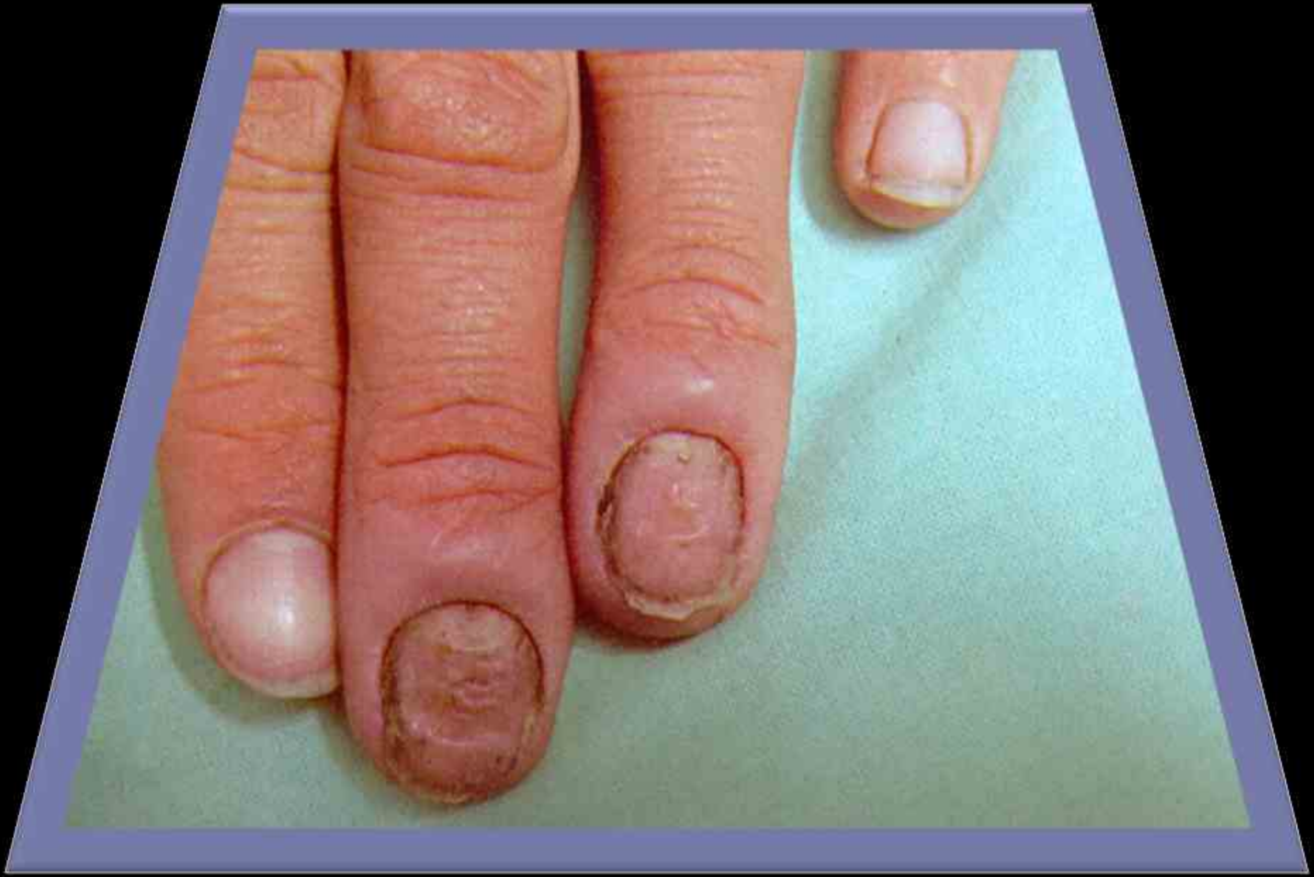
Spoon
Nails

Iron
Deficiency
Anemia



- ***Paronychia:***
  - Inflammation of the nail folds, occurs in:
    - Infections (bacterial, candidial).
    - Eczema.
    - Psoriasis.
    - Systemic lupus erythematosus.
- ***Leukonychia:***
  - White nails occurs in:
    - Arsenical poisoning.
    - Trauma.
    - Severe stress.
    - Liver cirrhosis.
    - Renal and cardiac diseases.
    - Vitiligo.

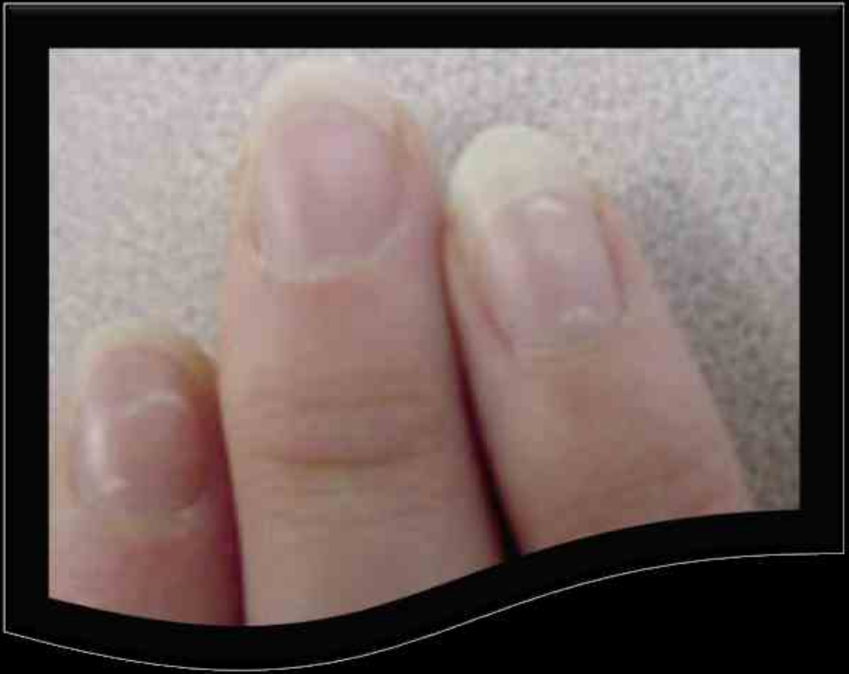
Paronychia



Leukonychia

Multiple whit spots

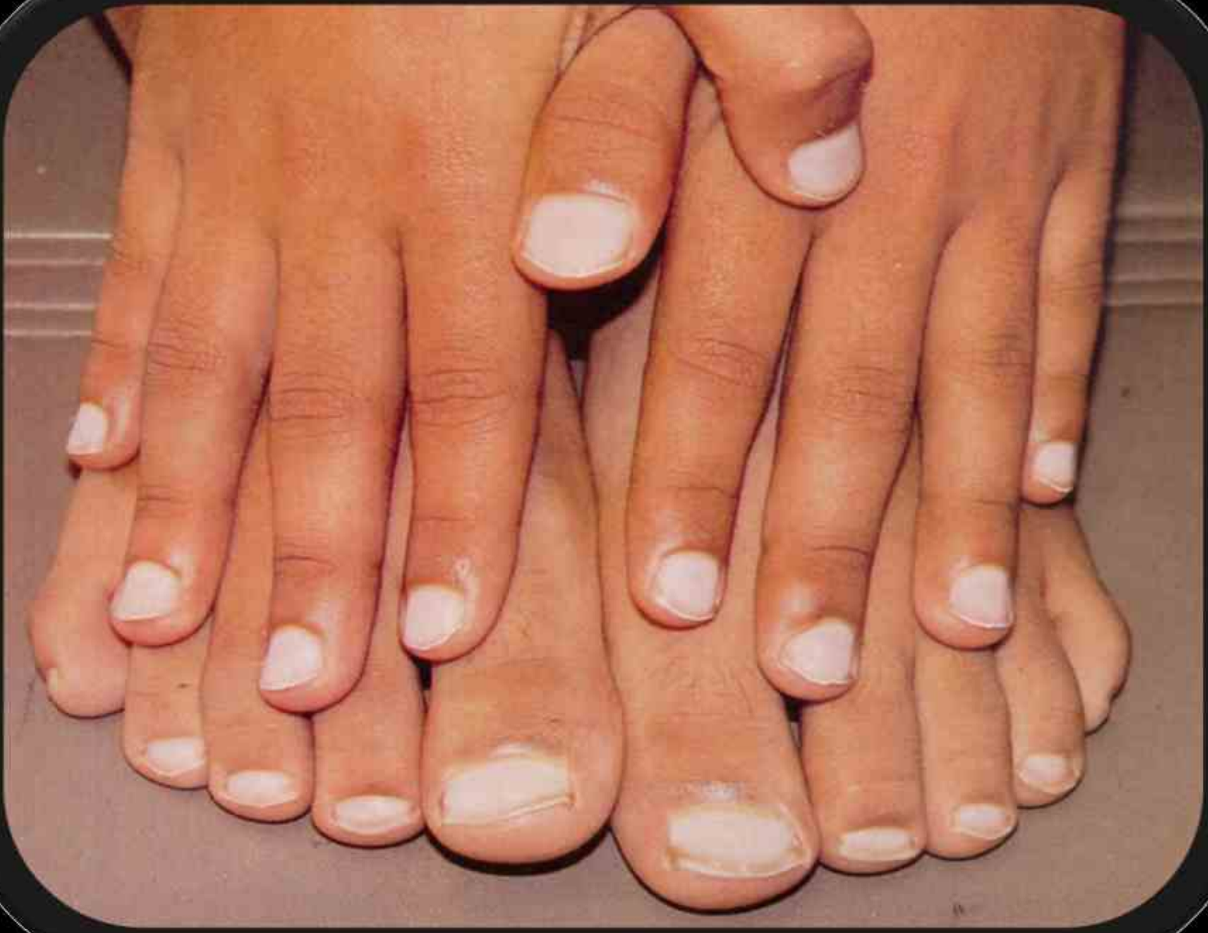
Liver disease



Diffuse Leukonychia

All nails

- ❈ *Keratodermas.*
- ❈ *Infections.*
- ❈ *Liver Ds.*
- ❈ *Drugs.*
- ❈ *Hereditary.*



- *Melanonychia:*
  - Black or dark brown discoloration of the nails, occurs in:
    - Trauma.
    - Melanoma.
    - Post-radiation.
    - Fungal infection.
    - Addison’s disease .
    - Vitamin B12 deficiency.
    - Drugs (gold, cytotoxic).
    - AIDS.

- *Splinter hemorrhage:*
  - Extravasation of blood in the nail bed, occurs in:
    - Subacute bacterial endocarditis.
    - Liver cirrhosis.
    - Scurvy.
    - trauma.

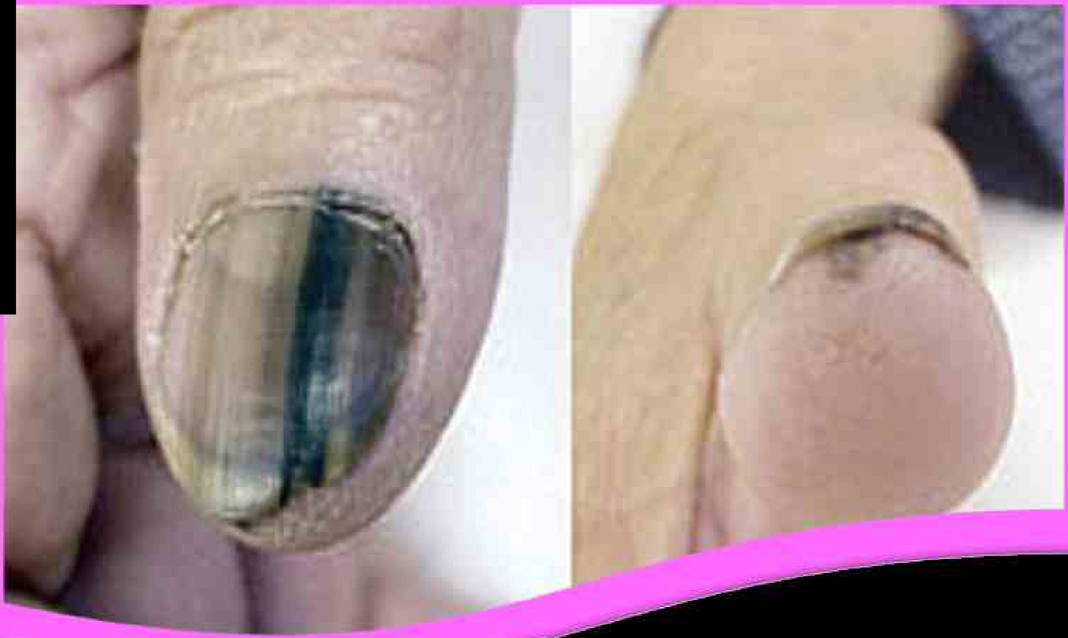
Melanonychia

Black pigmentation of nail

Partial or Complete

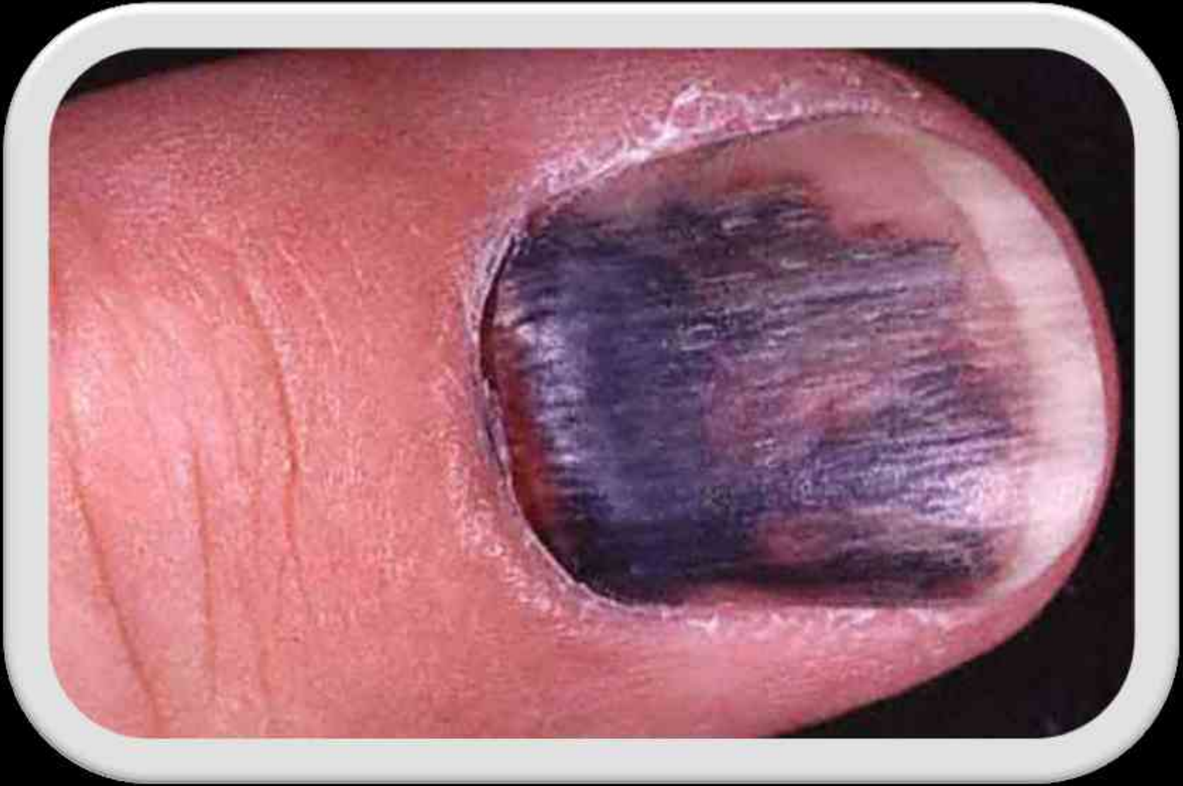
Malignant Melanoma

- ❉ Melanocytes hyperplasia.
- ❉ Nevus.
- ❉ MM



Sub-ungual Hemorrhage

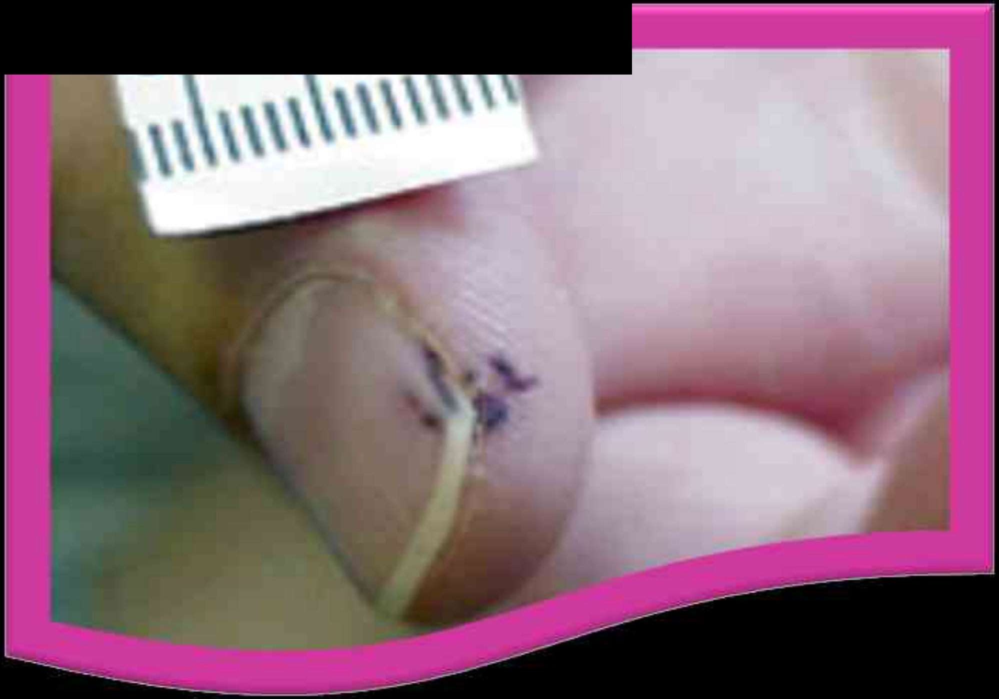
Diffuse: due to trauma



Splinter Hemorrhage

Nail plate and skin

- Trauma.
- Endocarditis.
- Skin Ds.



Nail involvement in skin diseases

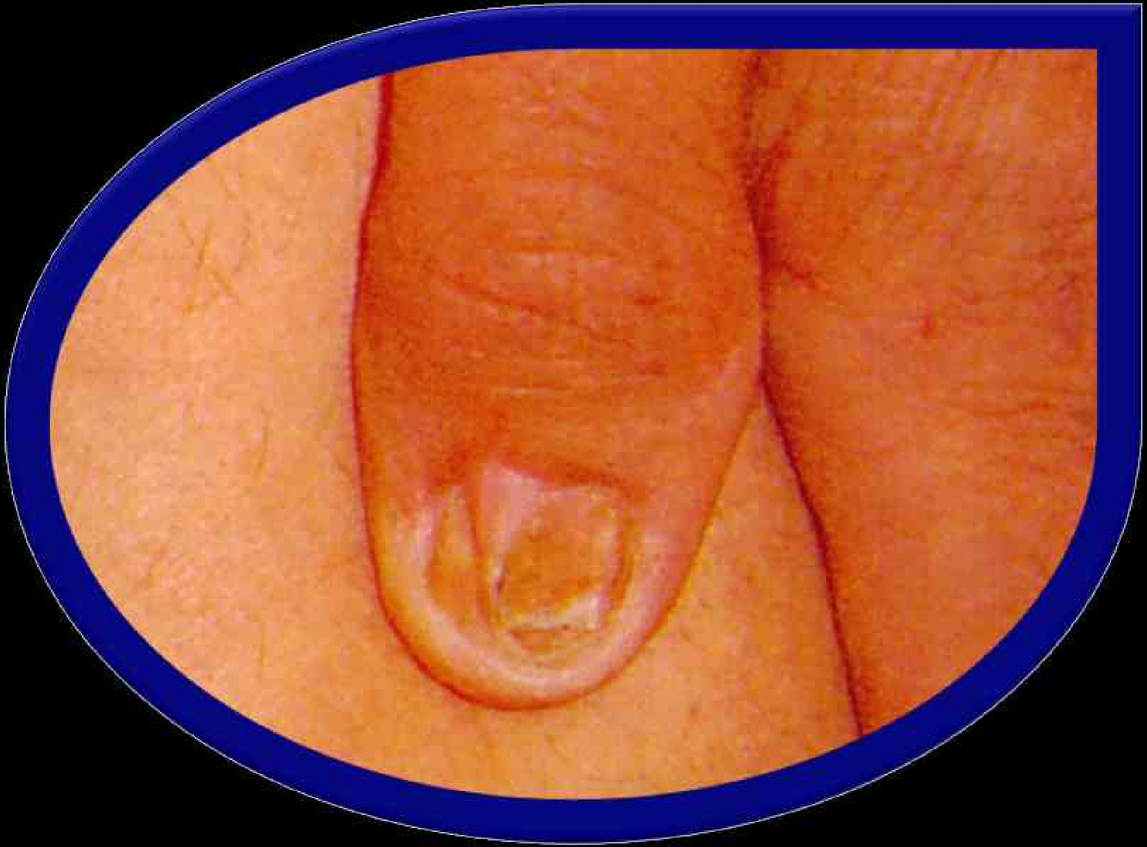
- ***Psoriasis:***
  - Pitting.
  - Onycholysis.
  - Yellowish discoloration.
  - Subungual hyperkeratosis.

- ***Lichen planus:***
  - Thinning.
  - Longitudinal grooving of nail plate.
  - Pterygium formation leading to nail dystrophy.

- ***Alopecia areata:***
  - Pitting: forming transverse rows on the surface of the nail plate.

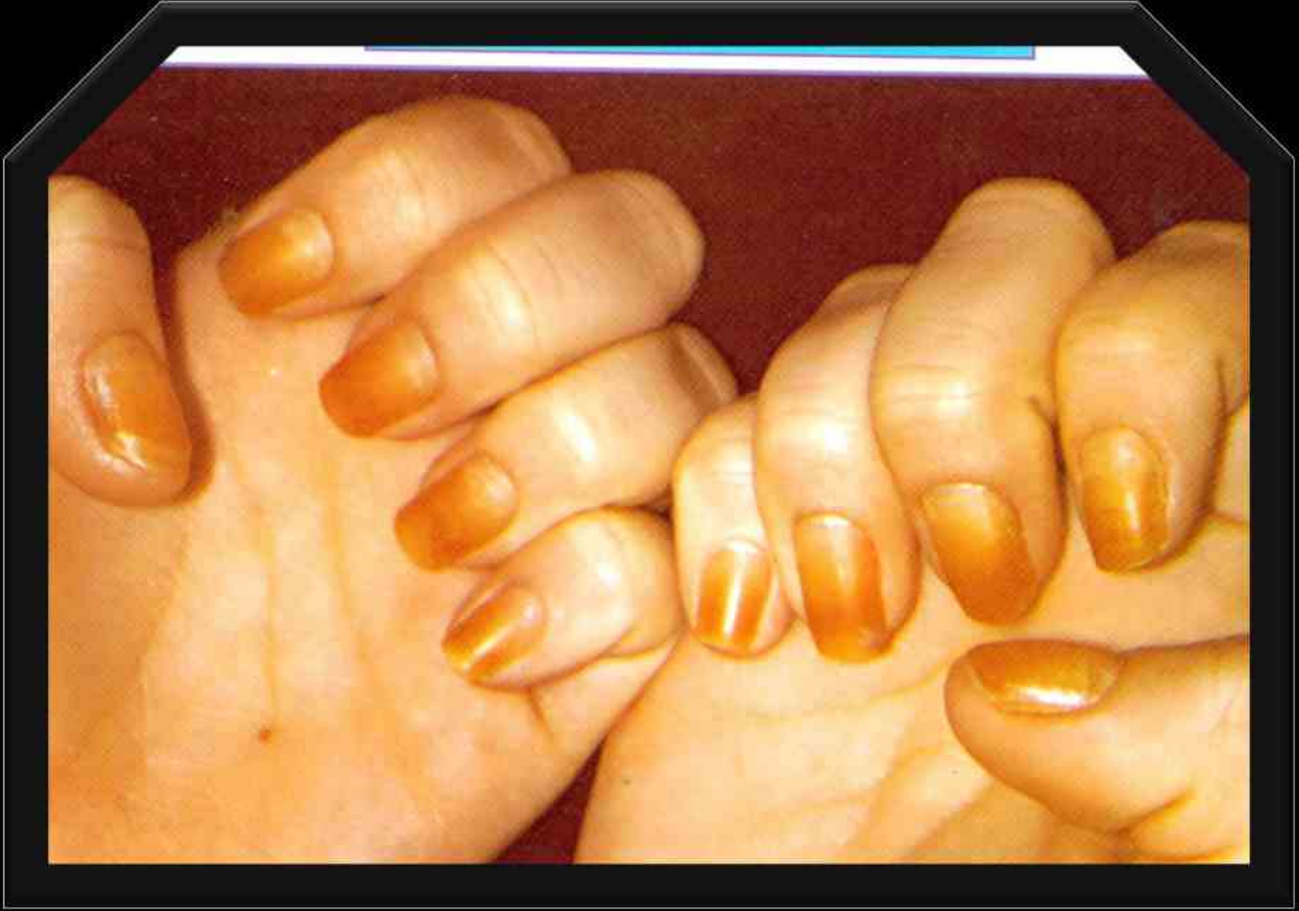
Pterygium

Lichen planus



Yellow Nails Syndrome

- Lymphedema.
- Pulmonary Ds.
- Autoimmune Ds.
- Drugs.
- Immunodeficiency.
- Malignancies.



- *Pityriasis rubra pilaris:* nails are:
  - Thickened
  - Brittle. or
  - Striated.
- *Nail infections:*
  - Viral: Molluscum contagiosum, warts.
  - Bacterial:
    - Pseudomonas (green nail plate).
    - Proteus (brown nail plate).
    - Bacterial paronychia.

  - Fungal infections:
    - Candidiasis: candidial paronychia.
    - Dermatophytic: onychomycosis:
      - Punctate leukonychias.
      - Onycholysis.
      - Subungual hyperkeratosis.
      - Melanonychia (T. rubrum).

HAIR DISORDERS

- Hair has no vital function in humans, yet its psychological functions are extremely important. Hair is present all over the body except the palms, soles, glans and prepuce.

Types of hair

- **Lanugo hair (prenatal).** Fine hair, unmedullated, unpigmented usually shed in utero.
- **Vellus hair (postnatal).** Similar to lanugo, seldom exceeds 2 cm in length.
- **Terminal hair.** Long, coarse, medullated and pigmented.

Androgen dependent hair

- Pubic and axillary hair in both sexes.
- Facial, trunk and extremities in males only.
- The average scalp hair is about 100,000 hair and the average number of hair shed is 25-100/day. Scalp hair growth is about 0.35 mm/day (1 cm/month).

- The average growth of scalp hair is 1cm/month.
- Androgen stimulate the growth of hair in:
  - Beard, mustache, chest, abdomen in males and axillae genital areas in both.
  - Scalp hair not required androgen for growth.
  - In genetically predisposed individuals androgen has inhibitory effect on growth of scalp hair leading to alopecia.
  - Source of androgen:
    - Testicular (♂).
    - Ovarian (♀).
    - Adrenocortical in both.

Types of hair

Lanugo hair	*Vellus hair*	*Terminal hair*
Fine and soft	Fine & soft	Long & coarse
Unmedullated	Unmedullated	Medullated
unpigmented Usaully		pigmented:
Covering the fetus prenataly.	Covering most body of youngs	❖ Situated in adults: Eye brows. Eye lashes. Scalp. Beard. Axillae. Pubes

Hair cycle

The hair doesn’t grow continuously, but each hair follicle has its own cyclic rhythm of growth and resting periods called stages which unsynchronized with other follicles

- *Anagen stage:*
  - The active stage.
  - Usaully of (2 – 8) years duration.
- *Catagen stage:*
  - transitional stage.
  - Take about 3 weeks.
- *Tellogen stage:*
  - The resting stage.
  - take about 3 months

- **Anagen (Growing Phase)**

  The growing phase lasts two to seven years and determines the length of our hair.

- **Catagen (Transition Phase)**

  This is the transitional stage that lasts about ten days – 21 days The hair follicle shrinks and detaches from the dermal papilla.

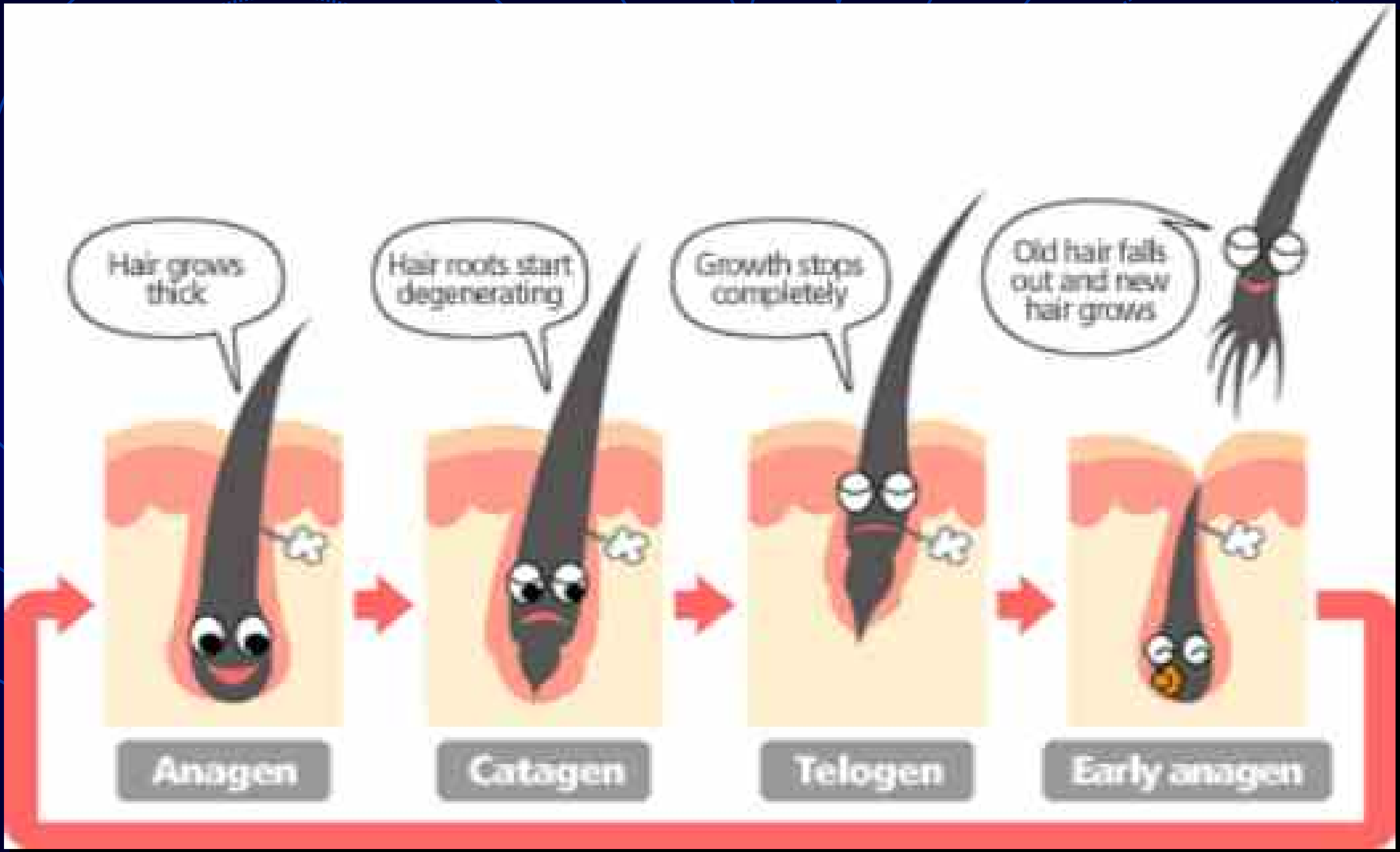
- **Telogen (Resting Phase)**

  This is the resting phase which lasts around three months. Around 10 -15 percent of hairs are in this phase. Whilst the old hair is resting, a new hair begins the growth phase.

- **Exogen (New Hair Phase)**

  This is part of the resting phase where the old hair sheds and a new hair continues to grow. Approximately 50 to 150 hairs can fall out daily, this is considered to be normal hair shedding.

If hairs enter the resting phase too early, excess shedding and noticeable thinning can occur.


Hair grows thick
Hair roots start degenerating
Growth stops completely
Old hair falls out and new hair grows
Anagen
Catagen
Telogen
Early anagen

Excessive hair growth

1. *Hypertrichosis:*

Excessive hair growth of non-androgenic pattern.

Causes:

1. Malignancies: some malignancies associated with growth of fine lanugo hair over a large area of the body e.g. GIT and bronchus tumor.
2. Drug induced: e.g.
    - Hydantoin.
    - minoxidil.
    - Streptomycin.
    - Psoralin (PUVA therapy).
3. others:
    - Trauma.
    - Topical corticosteroids.

2. Hirsutism:

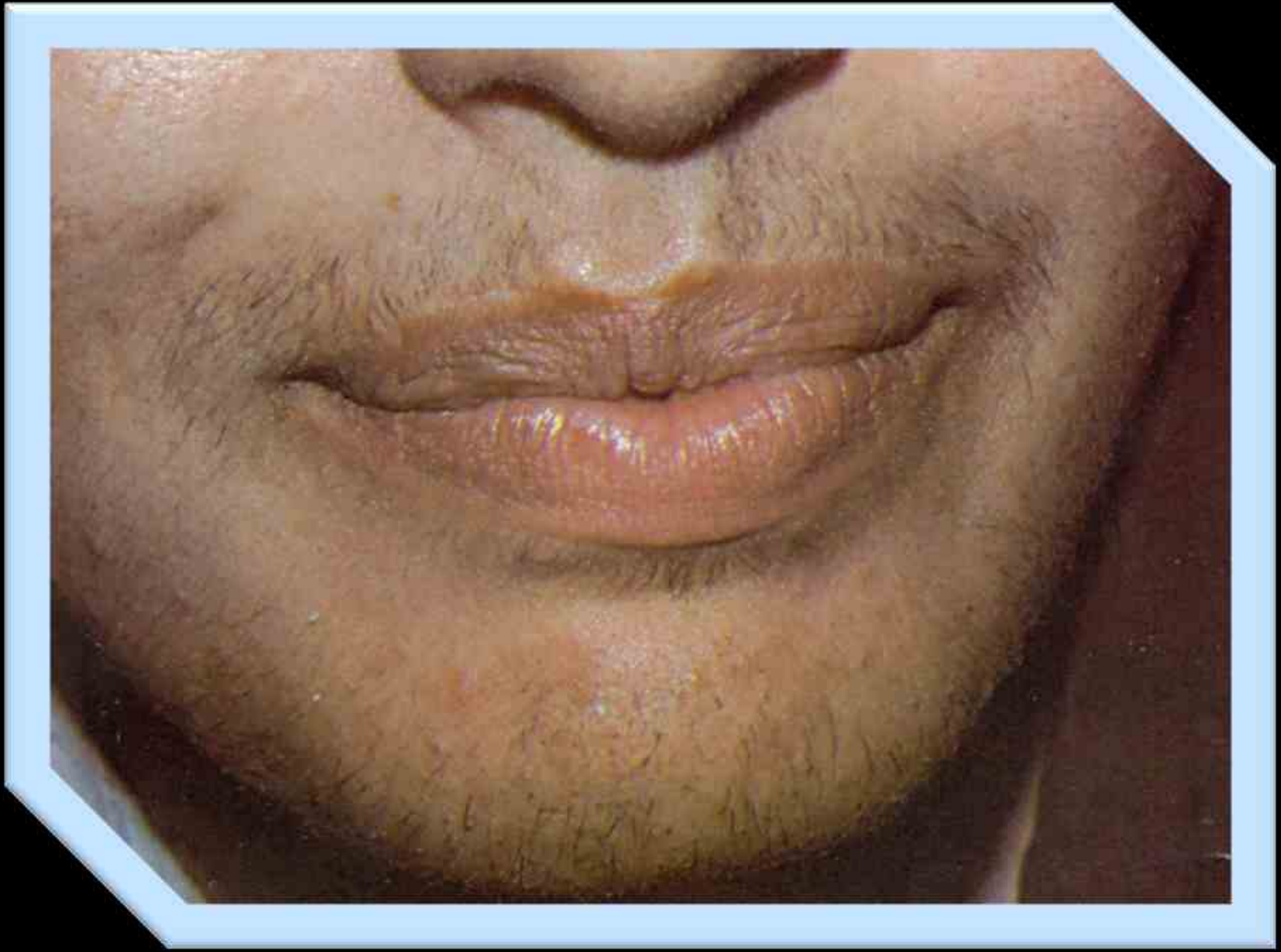
Excessive male- pattern hair growth in female.

Causes:

1. Idiopathic.
2. Ovarian causes e.g.
   - Polycystic ovarian syndrome.
   - Virilizing ovarian tumour.
3. Adrenal e.g.
   - Congenital adrenal hyperplasia.
   - Cushing’s syndrome.
   - Virilizing adrenal tumour.
4. Hyperprolactinemia.
5. Drug-induced:
   - Androgens.
   - Anabolic drugs.
   - Systemic corticosteroids.

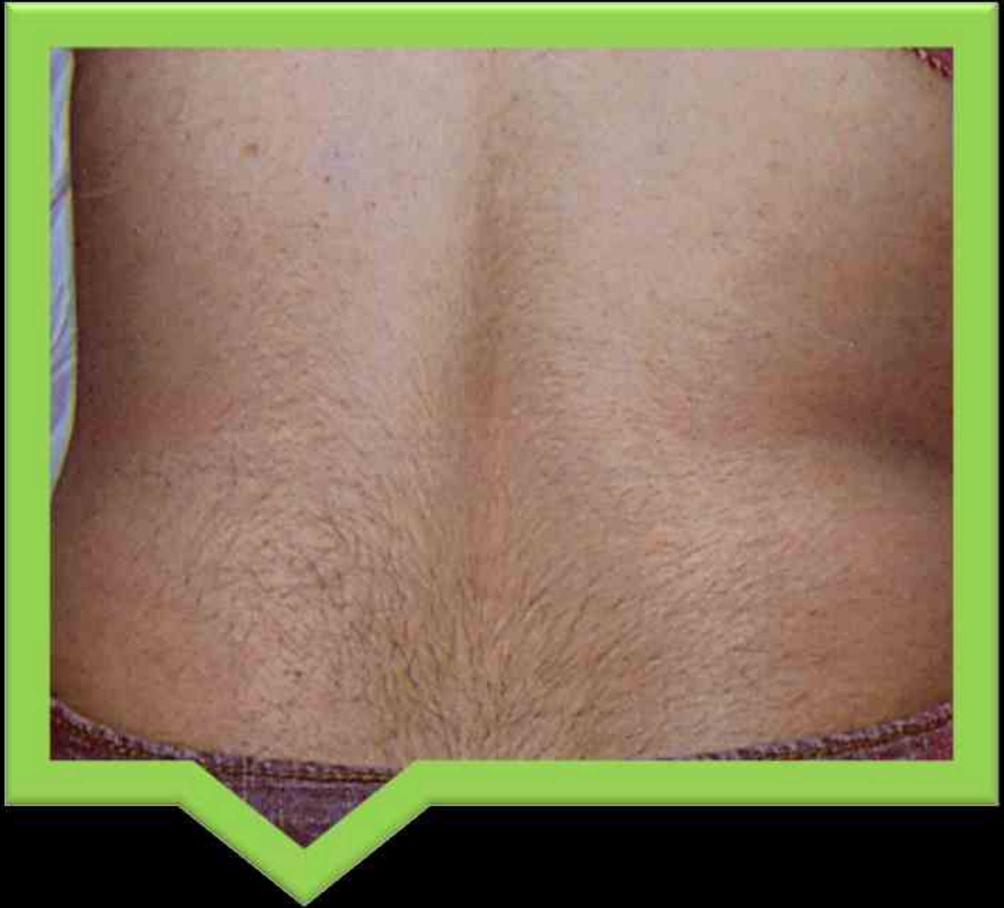
Hirsutism

Excessive male- pattern hair growth in females



Hirsutism

Excessive male- pattern hair growth in females



Alopecias

Alopecia means loss of hair. It may be cicatricial or non-cicatricial.

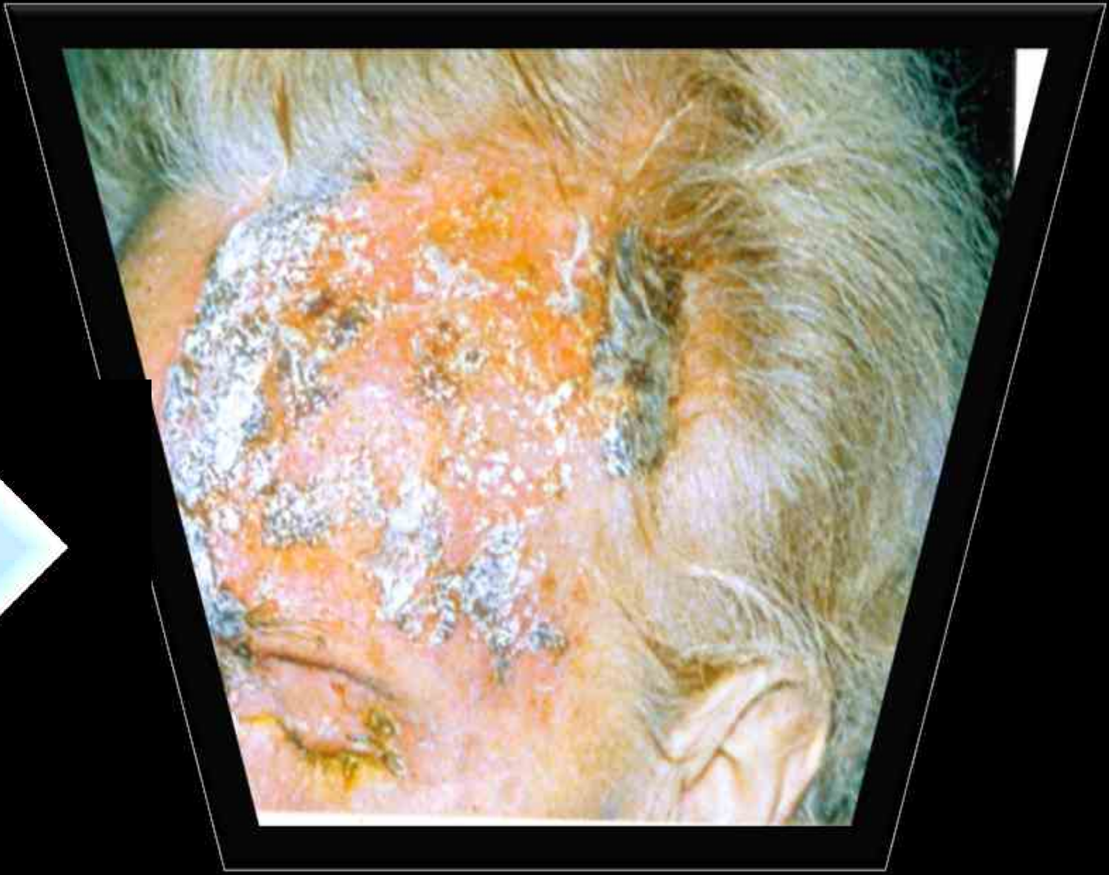
A- Cicatricial alopecia It results from destruction of hair follicles by scar tissue formed in the scalp. Cicatricial alopecia is a permanent condition and regrowth of hairs is not expected.

1. **Congenital**. A scarred area is present at birth due to a developmental defect.

2. . **Acquired**
   - *Trauma* (Mechanical, Thermal , Physical.
   - *Infection* (Pyogenic, Fungal,
   - *Collagen diseases (DLE)*
   - *Diseases of unknown etiology* Pseudopelade.

Cicatricial Alopecia

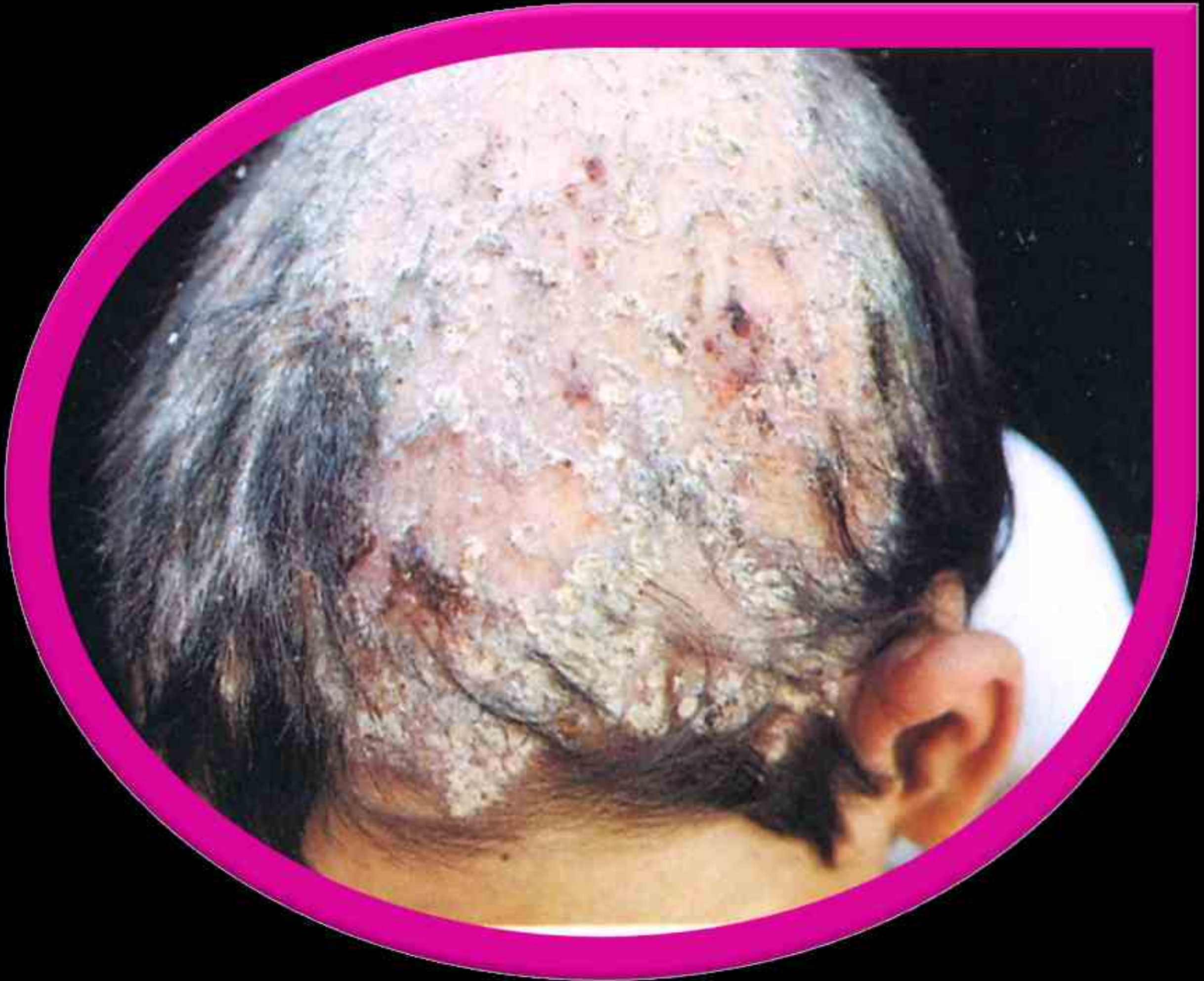
Herpes Zoster



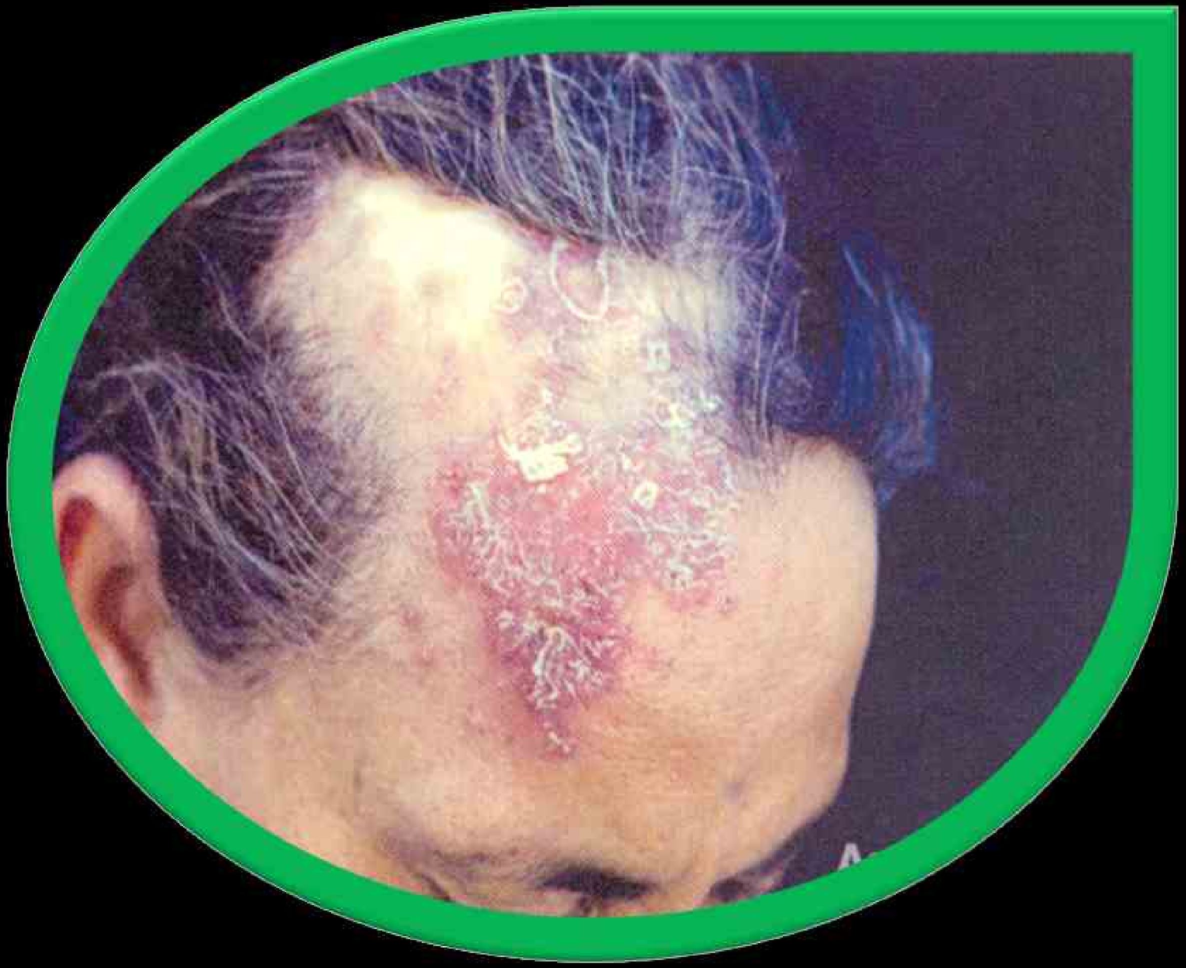
Cicatricial Alopecia

Tinea Capitis

Favus

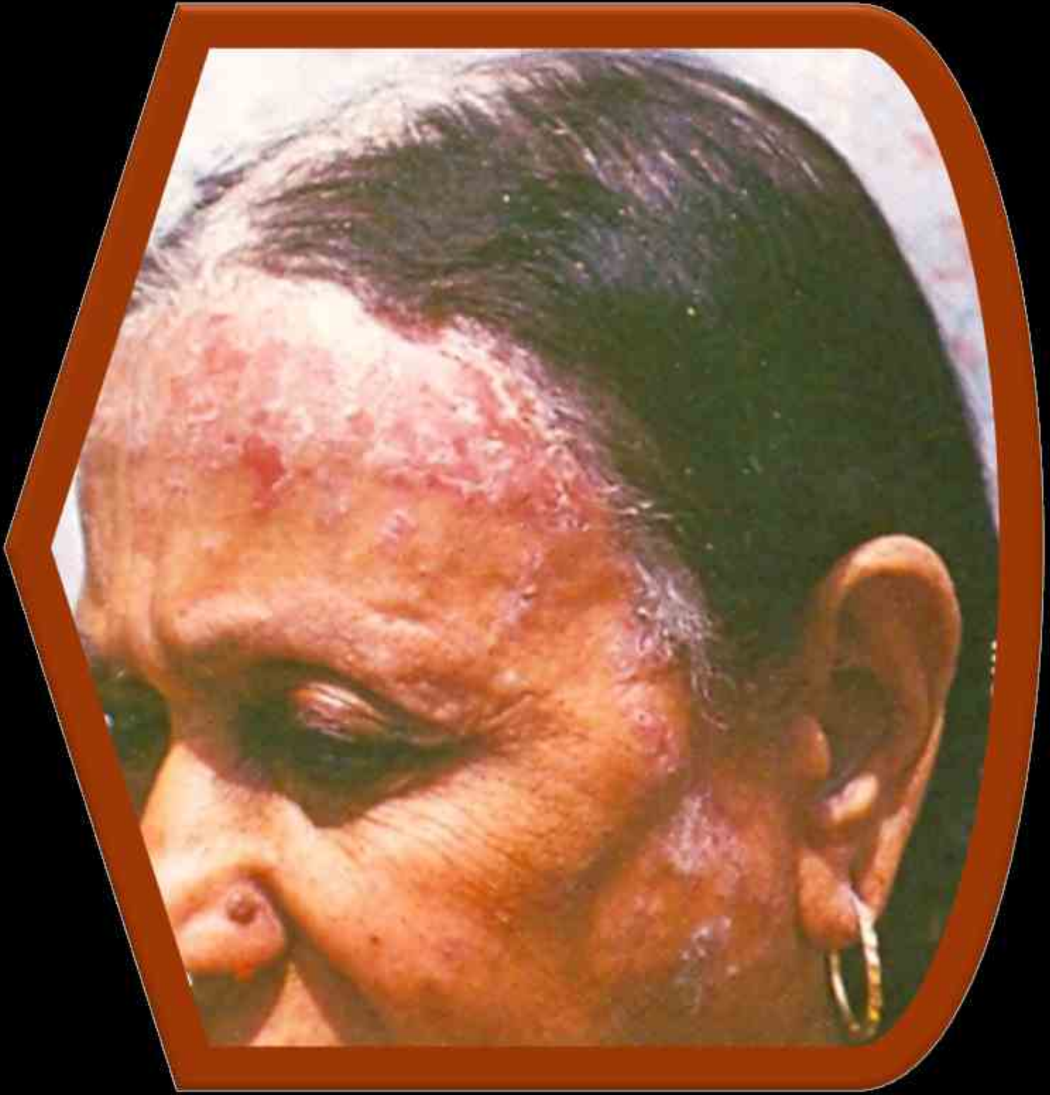


Cicatricial Alopecia

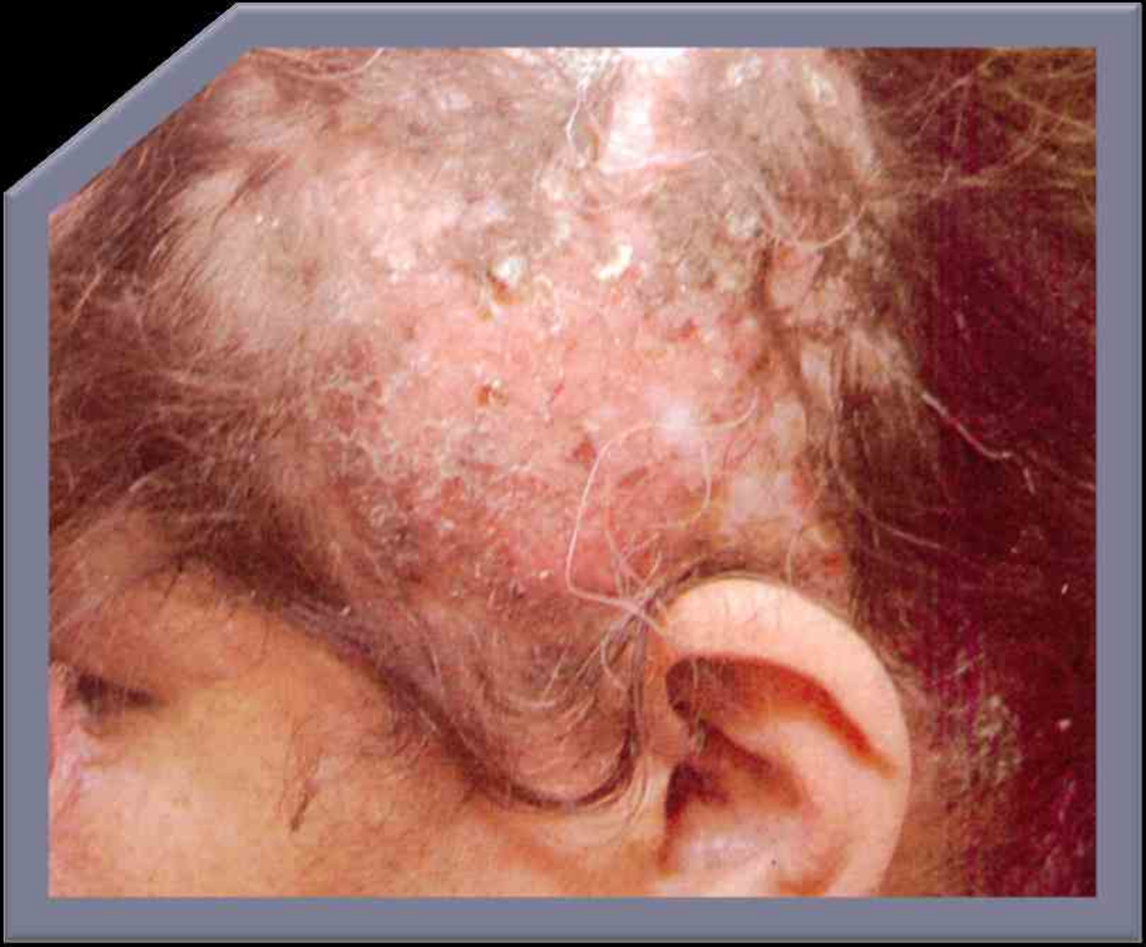


Lupus Vulgaris

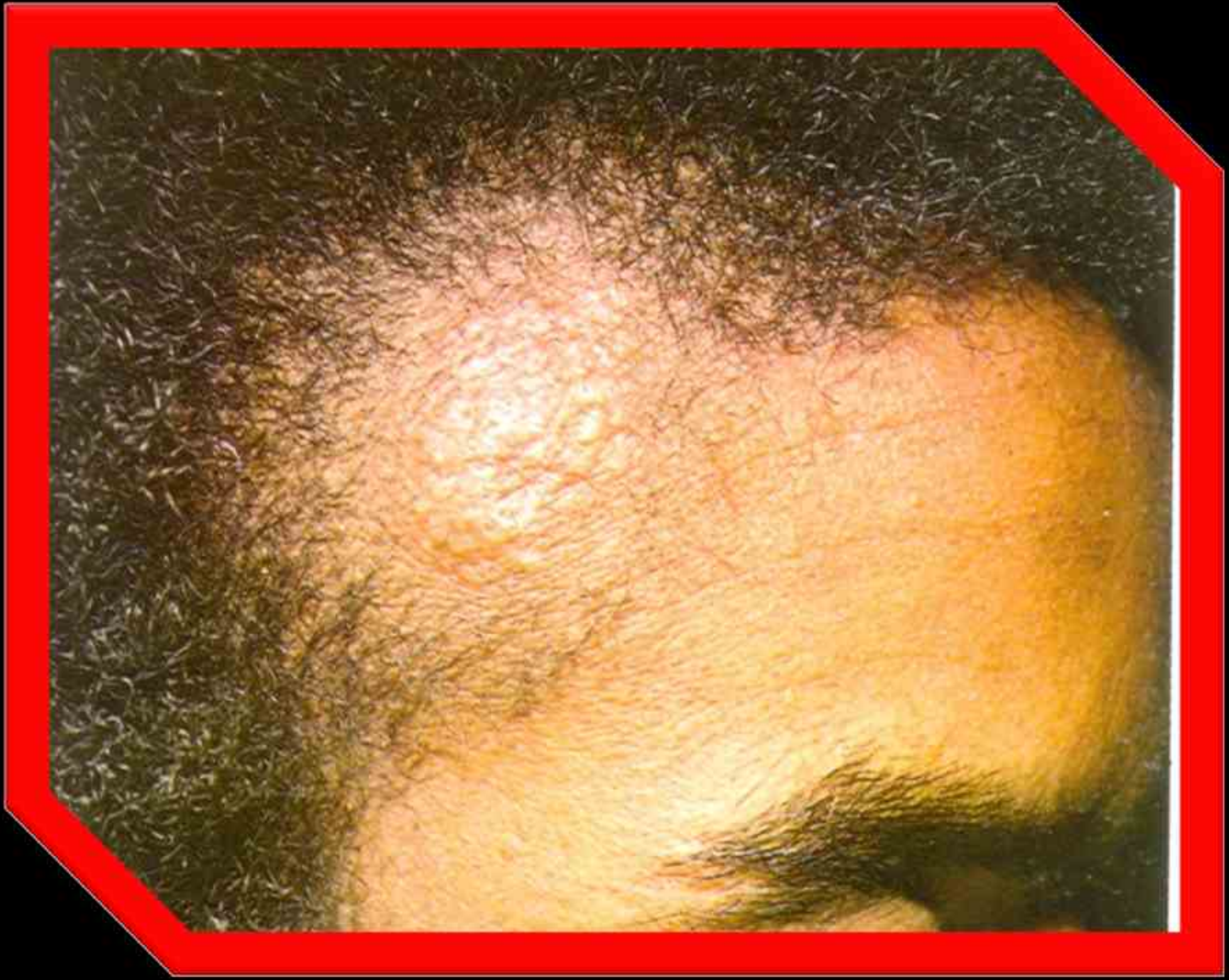
Cicatricial Alopecia
DLE



Cicatricial Alopecia
DLE



Cicatricial Alopecia
Sebaceous Nevus



B- Non-Cicatricial alopecia

1. Congenital

- Congenital atrichia. Due to failure of development of hair follicles.
- Congenital hypotrichia. The hair follicles are poorly developed.

2. Acquired

1- Circumscribed

- Alopecia areata.
- Infections e.g. tinea capitis, secondary syphilis (moth eaten alopecia).
- Traumatic (trichotillomania).

2- Diffuse

- Telogen effluvium (stress induced).
- Androgenetic alopecia.
- Drugs (thyroid antagonists, anticoagulants, arsenic, thallium salts).

Alopecia areata

- It is a common asymptomatic disease characterized by rapid (sudden) onset of hair loss in a sharply defined area.

Etiology

The etiology is unknown, but many factors appear to have a role:

  - **Genetic factors.** Positive family history in about 20%.
  - **Immunological factors**. supported by the association with other autoimmune diseases.
  - **Emotional stress.** May be a precipitating factor.

Clinical picture

- Alopecia areata occurs mostly in patients below 40 years age, and both sexes are equally affected. It is characterized by rapid and complete loss of hair in one or more circumscribed, round or oval patches, usually on the scalp (60%), bearded area, eyebrows, eyelashes, and less commonly, on other hairy areas of the body. Exclamation mark hairs "thin proximally and thick distally and can be easily pulled-out" may be present around the patch indicating progression of the disease.
- The course of the disease is unpredictable. There is a tendency to complete regrowth (4-6 months up to 2 years) but some

Clinical types

- **Alopecia localisata.** Described before.
- **Alopecia totalis.** Means total loss of scalp hair
- **Alopecia universalis**. Means loss of all body hair.

Treatment

Psychological assurance is needed.

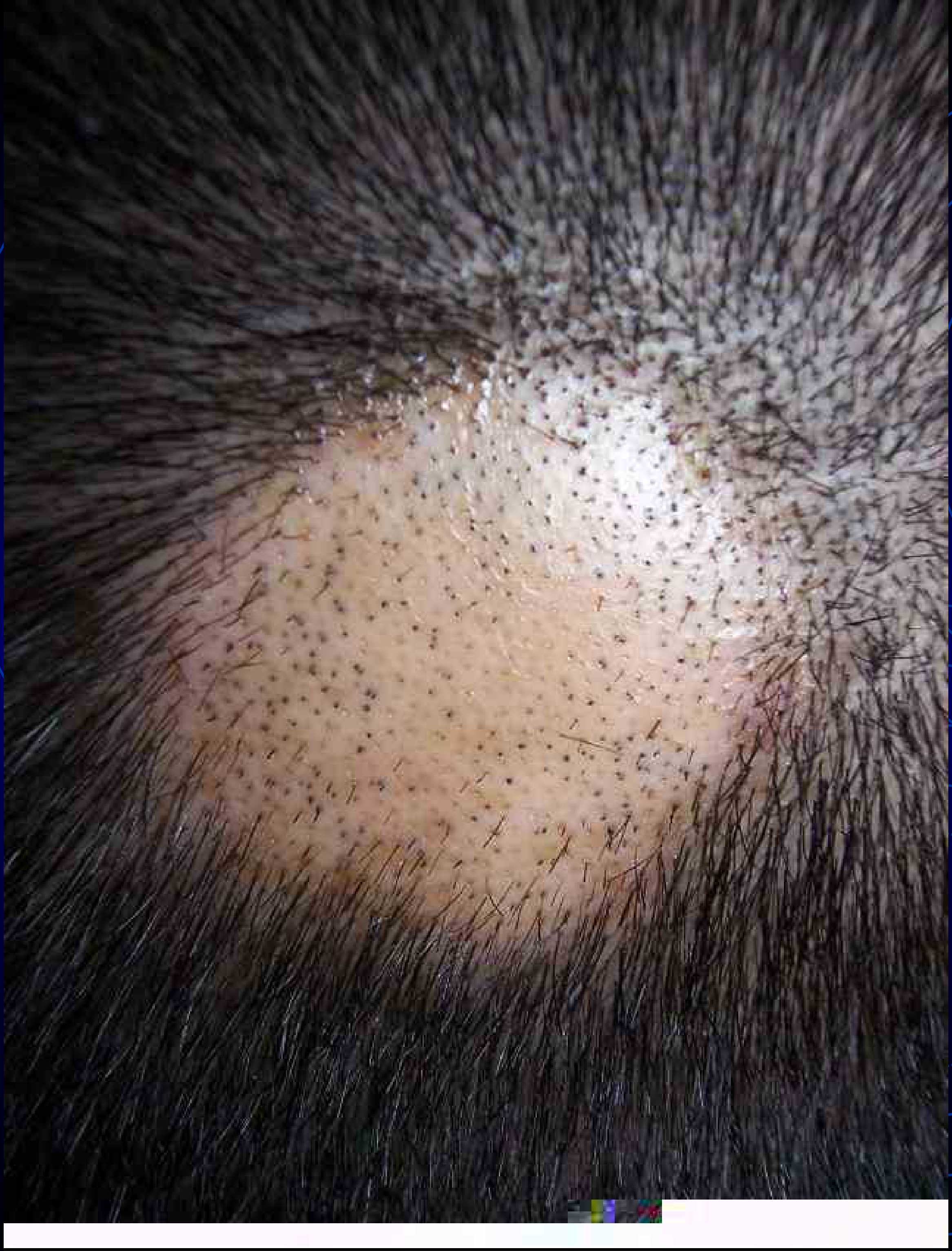
1- Topical treatment

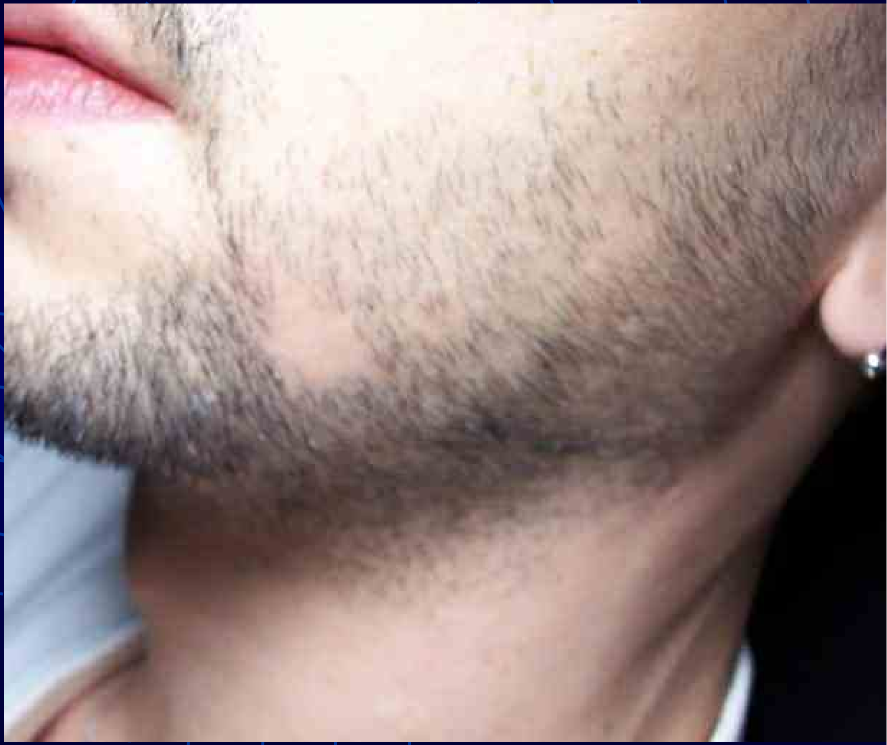
- a- Topical steroids.
- b- Local irritants e.g. dithranol, phenol.
- c- Minoxidil 5% “Regaine”.
- d- Topical immunotherapy e.g. dinitrochlorobenzene (DNCB).
- e- Topical cyclosporine.

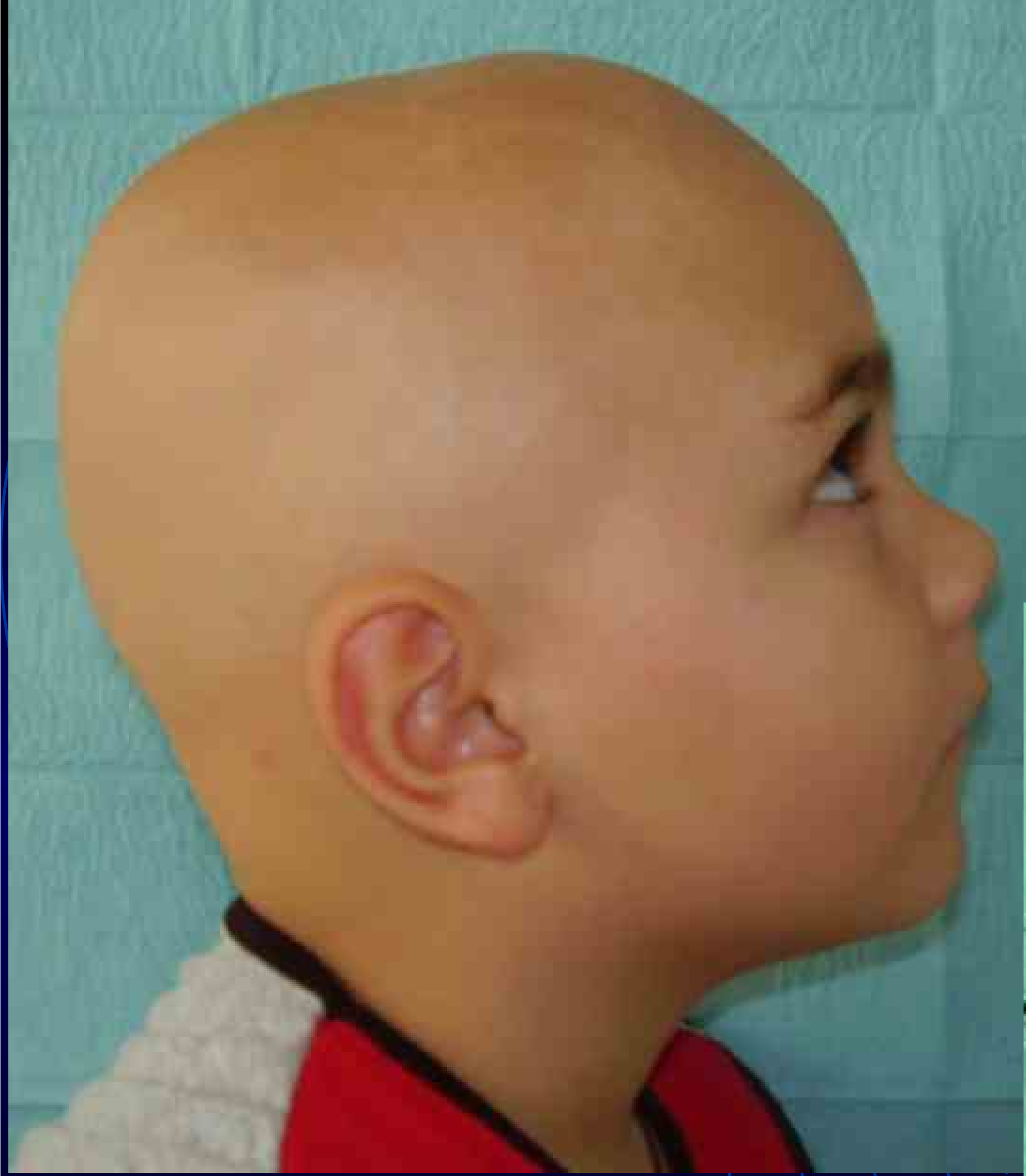
2- Intracutaneous injection of steroids

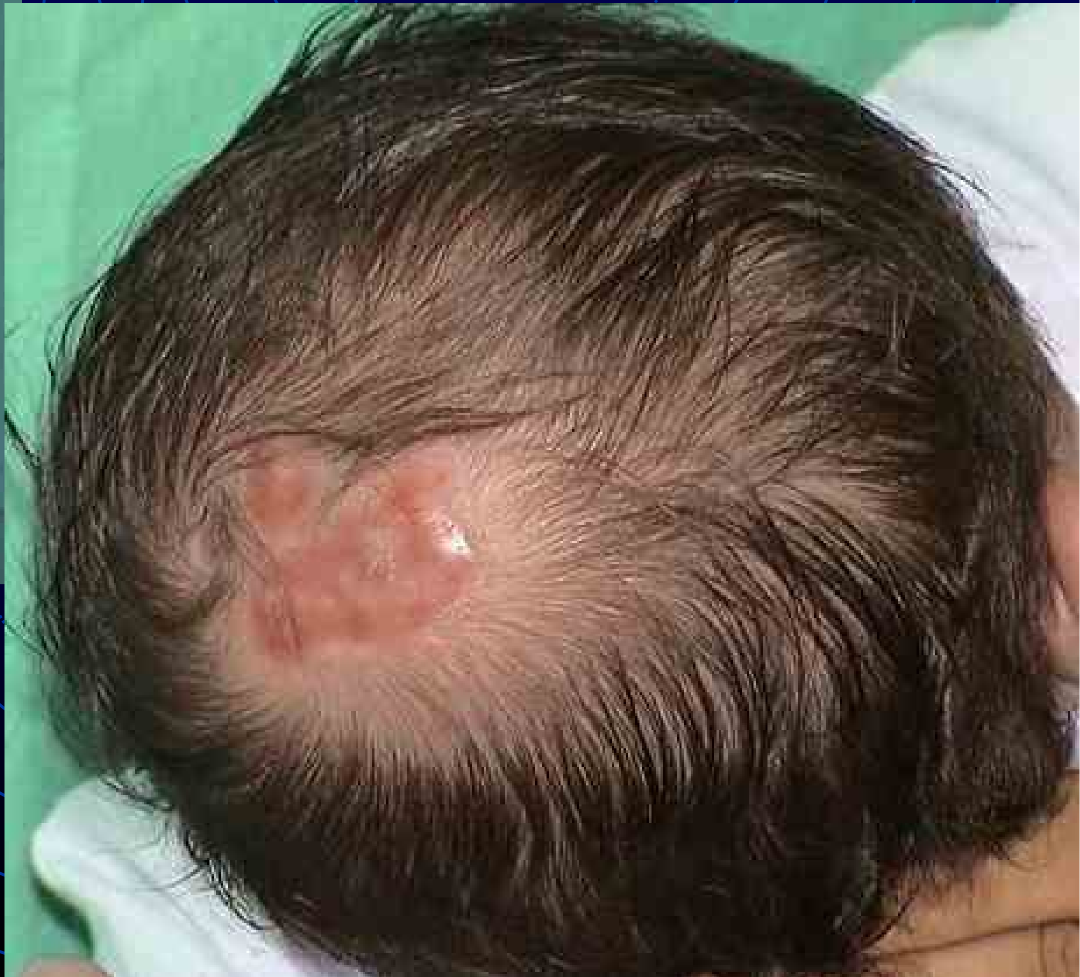
3- Systemic treatment

- Systemic steroids, can lead to hair growth but hair may be lost when the treatment is stopped.
- b- Systemic cyclosporine.
- c- Photochemotherapy (PUVA).

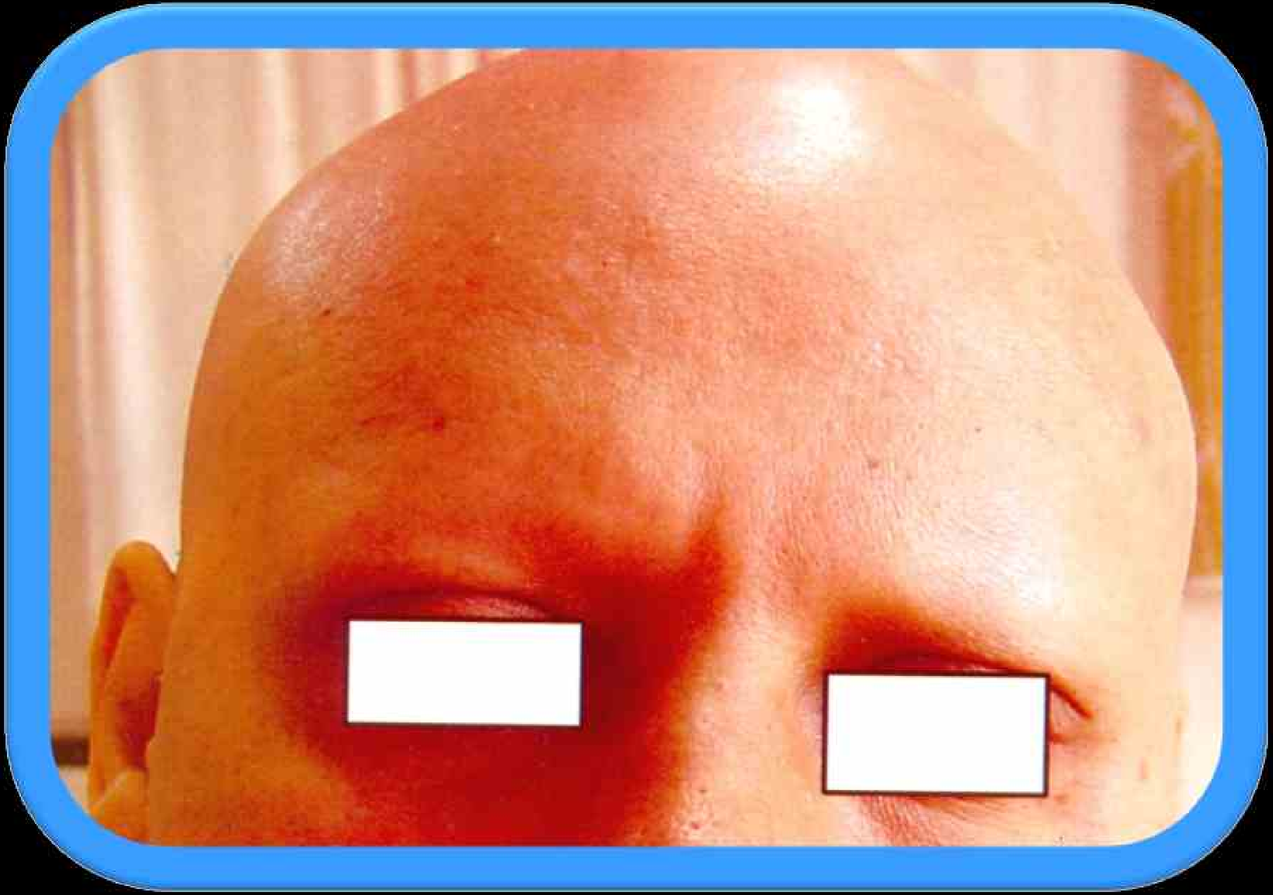








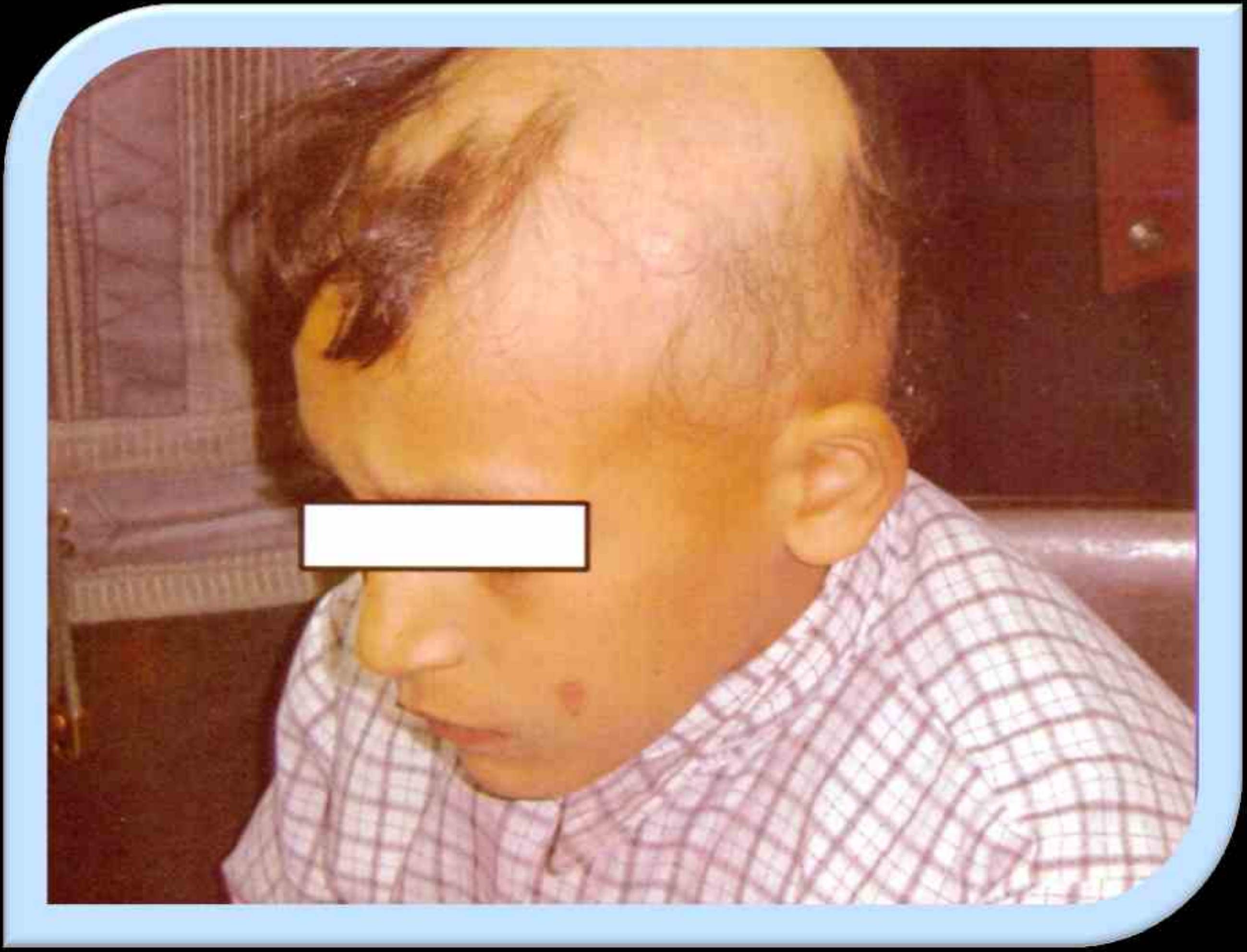
Alopecia Universalis



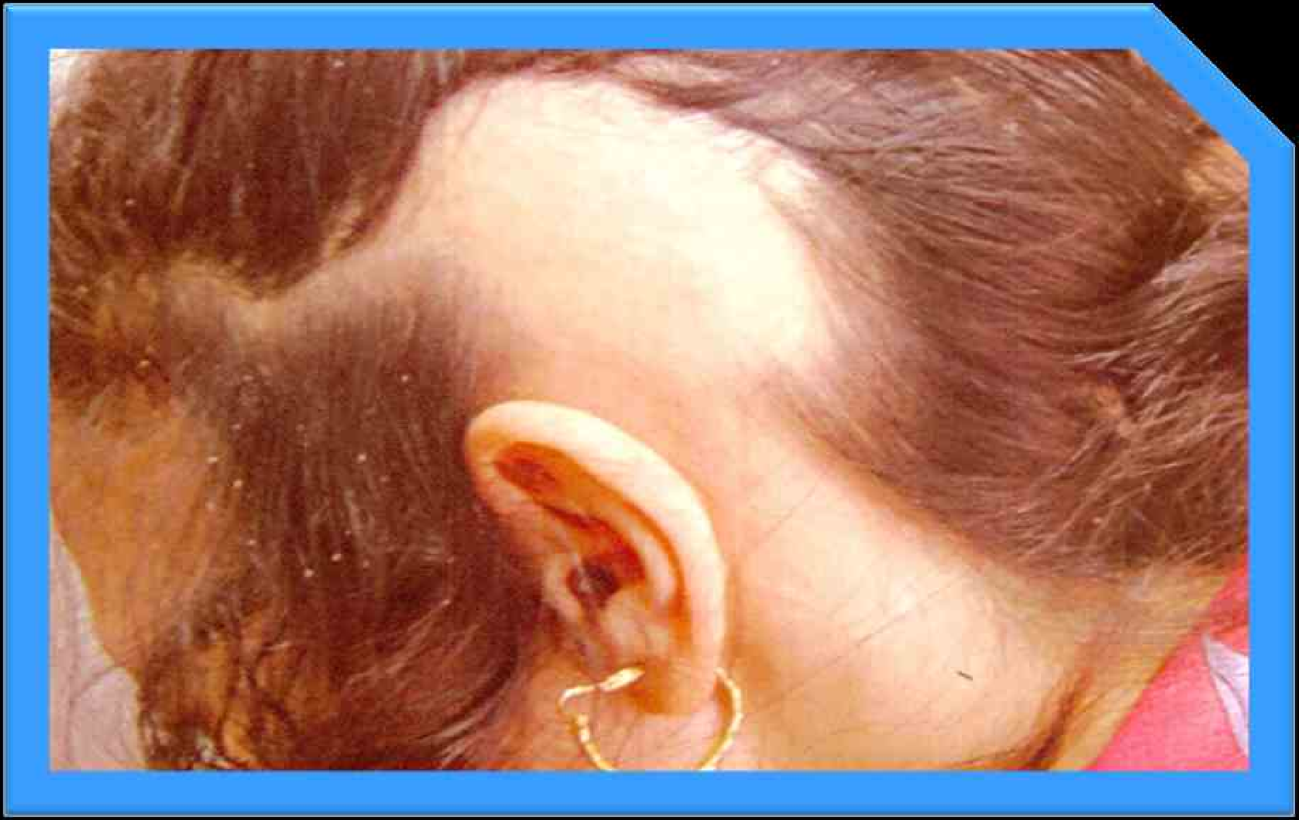
Alopecia Areata

Ophiasis

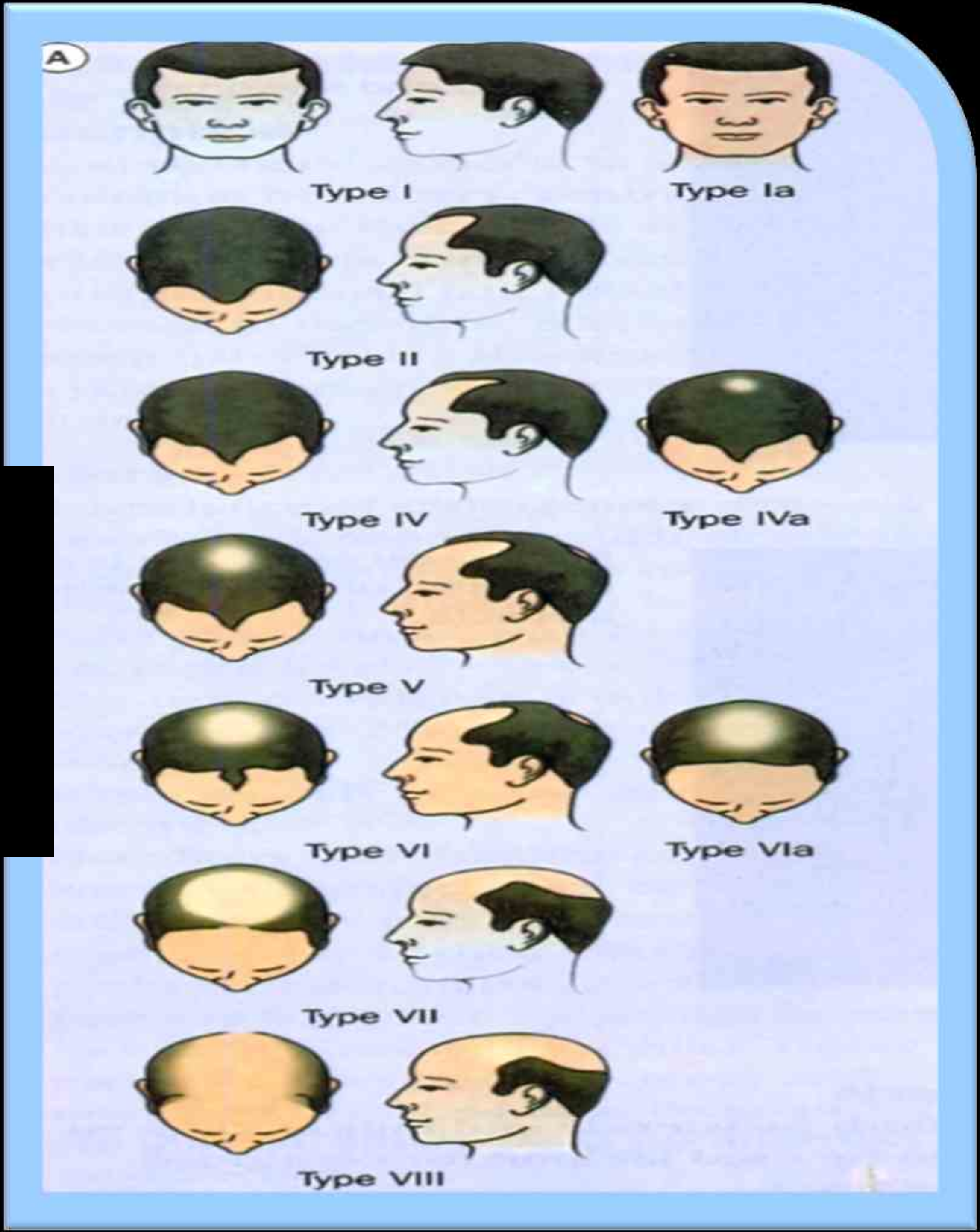
Bad Prognosis



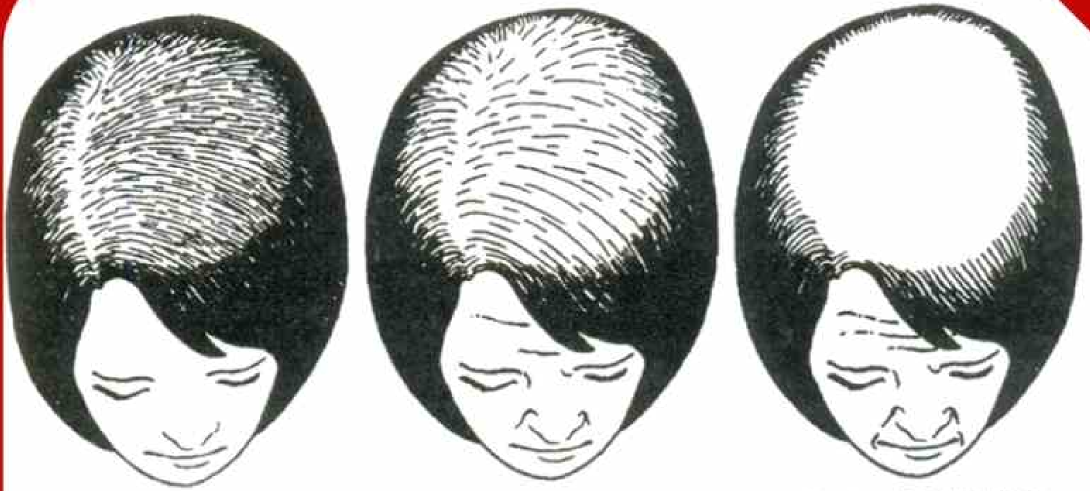
Alopecia Areata
Ophiasis



AGA


A
Type I
Type Ia
Type II
Type IV
Type IVa
Type V
Type VI
Type VIa
Type VII
Type VIII

AGA in females



Evolution of the female type of androgenetic alopecia. (From Montagna W, Parakkal PF (1974) *The Structure and Function of Skin*, 3rd ed. New York: Academic Press.)

Telogen effluvium

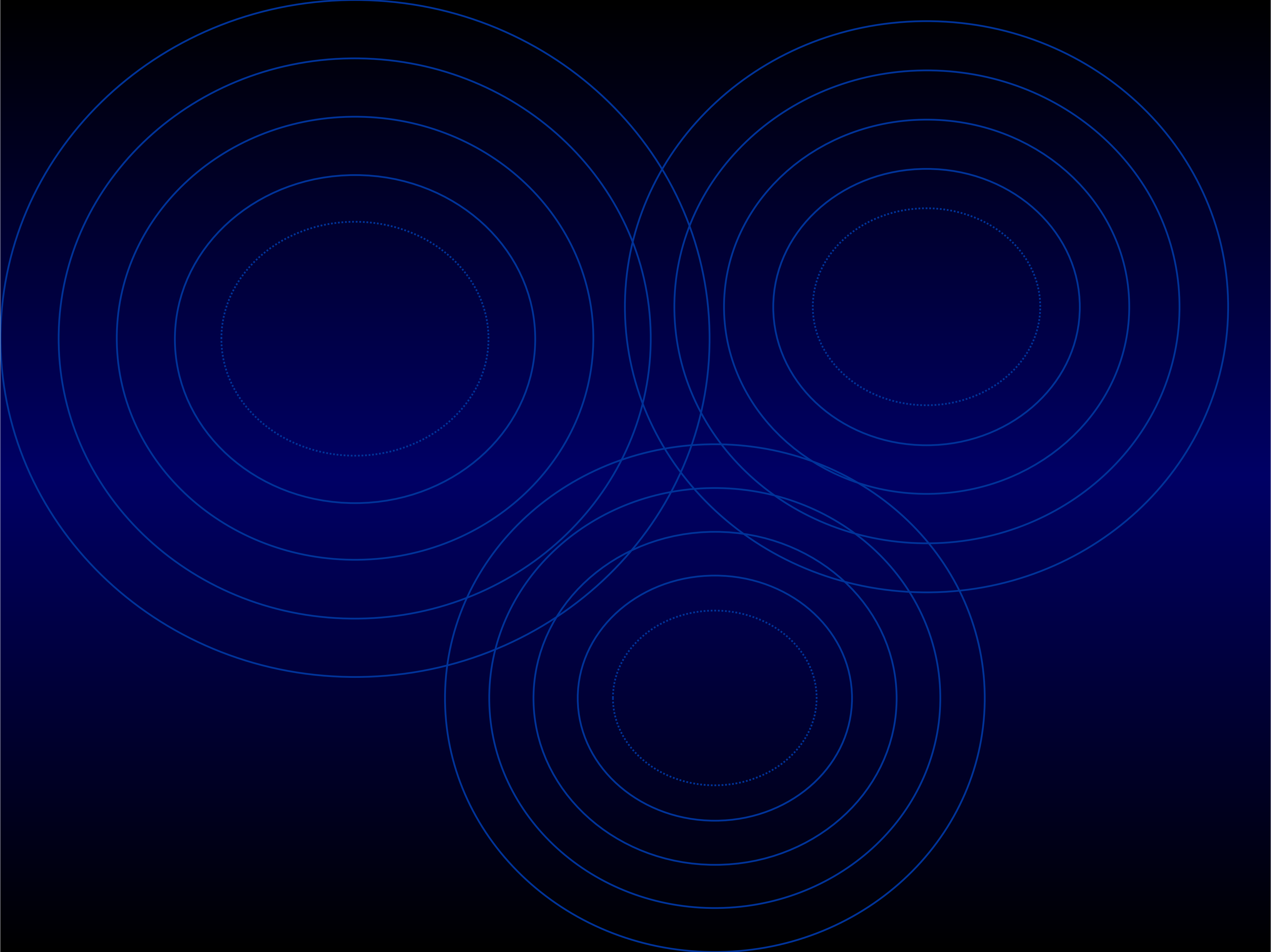
Following stress conditions many anagen hair follicles enter prematurely into telogen with excessive loss of normal hairs.

Causes

- - Labour
- Acute blood loss and surgical operations
- - High fever
- Emotional stress
- Crash diet (inadequate protein diet).

Clinical picture

Diffuse shedding of hair occurs 1.5 to 4 months after exposure to stressful event. All the shed hairs are in the telogen phase. Usually no more than 50% of the hairs are affected. The prognosis is good as complete



Pigmentary disorders

- Skin color is due to mixture of pigments; melanin, haemoglobin and carotene
- Excess of melanin is called **hypermelanosis (hyperpigmentation)** as **melasma,** while deficient or decrease of melanin is called **hypomelanosis (hypopigmentation) as vitiligo.**
- **Hyperpigmentation:** due to melanin or non melanin pigment in the skin.

Etiology: **1-** genetic; freckles

2- endocrine; melasma, Addison disease

3- nutritunal; carotenemia, Pellagra

4- postinflammatory; post acne, post LP.

5- drugs: minocycline, psoralens

6-ultraviolet radiation.

Hypomelanosis (leukoderma)

reduction in melanin or absence

- **Etiology**

1- vitiligo and albinism

2- chemical; phenol and drugs; hydroquinone.

3 – post inflammatory; post burn, pit versicolor, psoraisis, pit alpa ,

4- hypo and hyperthyroidism

Vitiligo

It is an acquired loss of pigment of the skin. Vitiligo usually begins in childhood and affects both sexes. It occurs in about 1 % of the world's population.

Etiology The etiology is still unknown.

1- **autoimmune theory:** devielop autoantibodies against melanocytes which cause destruction of melanocytes. This theory is proved by the association of vitiligo with autoimmune diseases (thyroid dis. , Alop. Areata, Pernicious aneamia, addison dis. , Diabetes mellatus)

2- **neurogenic theory:** melanocytotoxic substances released from nerve endings cause inhibition of melanocytes.

3- **melanocytes self destruction :** due to a defect of natural protective mechanism.

Clinical picture

The disease is manifested by depigmented white patches surrounded by a normal or a hyperpigmented border. The hairs in the vitiliginous areas usually become white also. The lesions

Clinical classification

1- localized: focal, segmental

2- generalized: vitiligo vulgaris, acrofacial, vitiligo universalis.

- *Focal vitiligo* is usually a solitary macule or a few scattered macules in 1 area, most commonly in the distribution of the trigeminal nerve.
- *Segmental vitiligo:* presents as unilateral macules in a dermatomal distribution. This type tends to have an early age of onset.
- *Acrofacial vitiligo* presents as depigmentation of the distal fingers and periorificial areas.
- *Generalized vitiligo* is also termed vitiligo vulgaris. This is the most common pattern. Depigmented patches are widely and usually symmetrically distributed.
- *Universal vitiligo* presents as depigmented macules and patches over most of the body

Diff Diagnosis: **pit alpa , Pit versicolor, Postinflamatory hypopigmentation**

Treatment:

The treatment of vitiligo is generally unsatisfactory.

-**phototherapy:** (1) PUVA: oral or topical Psoralen followed by exposure to Ultra Violet light type A

(2) Narrow Band UVB (NBUVB) and excimer light

-**Medical treatment:** (1) Steroids; topical and systemic, (2)Immunomodulators (tacrolimus and pimecrolimus ointment (3)Antioxidants.

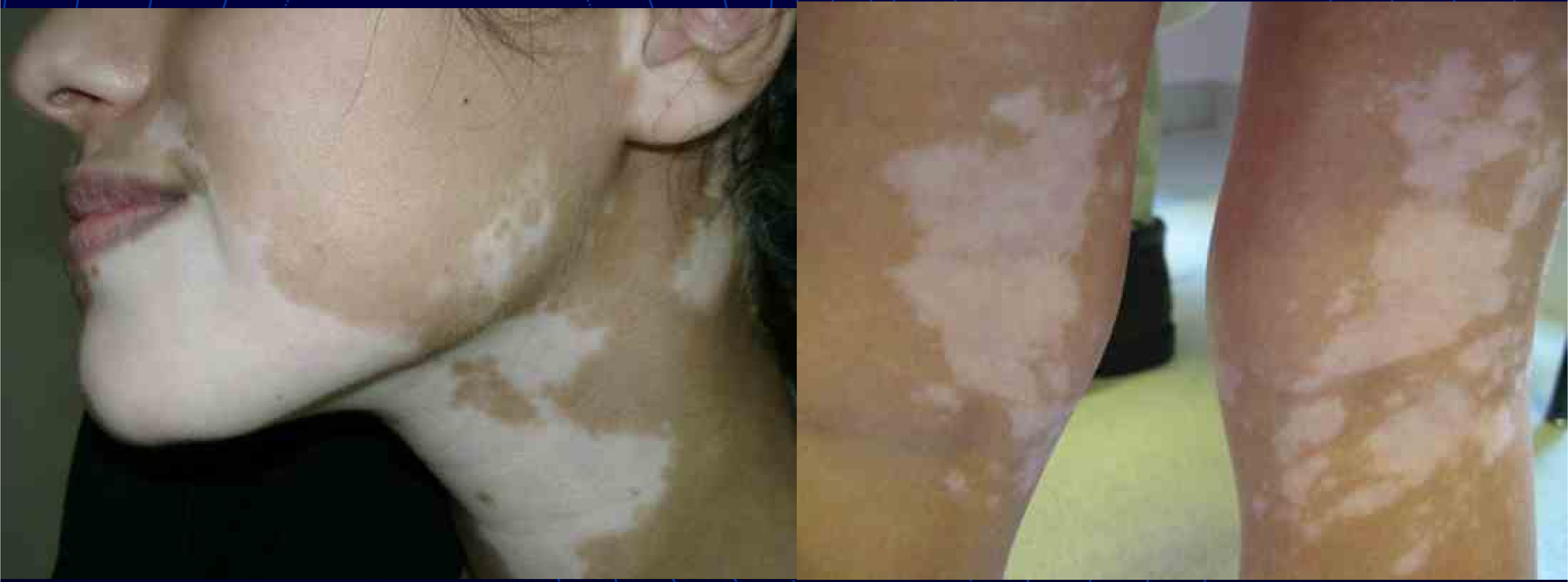
- **Surgical treatment:** (1) Punch grafting from normal to diseased site.

(2) Tissue culture of melanocytes.

- **Depigmentation:** Monobenzyl ether of hydroquinone in pts with generalized vitiligo that involves more than 60% of the body surfuce

-**Camouflage:** Skin-colored materials to hide affected

Vitiligo



remember,.. it could always be worse....

thankyou